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LVI.

HISTOLOGIC PATHOLOGY OF THE ACCESSORY SINUSES.*

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In presenting this subject, it is the purpose of the writer to illustrate histologic specimens of tissues removed from cases operated upon for the relief of chronic sinusitis and to draw some practical conclusions as to the etiology and treatment of these affections. It is intended to bring out most prominently the minute pathology in cases of chronic sinusitis, suppurative and nonsuppurative, in which the symptoms of asthma were prominent.

The work is based upon the examination of a large number of specimens from one hundred and forty cases operated upon

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for chronic sinusitis, and typical specimens have been selected for this publication. The clinical data or histories, course and final outcome of these cases, would aid a great deal in this report, but for lack of time and space they will be omitted. Suffice it to say that the study of minute pathology has brought a wealth of information to the writer, and the study of these specimens, in connection with the clinical phase of the subject alone, is of considerable practical value.

In the study of microscopic specimens of middle turbinates and ethmoid curettements of the cases of asthma, whether in suppurative or nonsuppurative cases, it has been noted that the bone changes are quite uniform in showing the formation of large areas of rarefaction. (Figs. 7, 15 and 16.) However, we have very little evidence of inflammatory processes about these rarefied areas. The tissue covering the bone, nevertheless, shows pathologically considerable variation. In some cases there is a great deal of inflammatory edema, with very little inflammation and round cell infiltration. (Fig. 3.) In others the round cell infiltration and old connective tissue predominate (Figs. 4 and 6), while in still other cases the glands are distended and increased in number (Figs. 2 and 3), or the glandular structures have undergone a myxomatous degeneration. (Fig. 3.) The epithelium varies considerably in its structure; at times it is very markedly thickened columnar (Fig. 14), as well as stratified. (Fig. 13.) Cilia can very seldom be discerned, and in some cases there are areas where the epithelium is entirely wanting.

In regard to the microscopic examination of cysts in the middle turbinates, they resemble considerably the changes as mentioned above, except that the lining epithelium of the cavity and the subepithelial structure appear to have characteristic differentiation. The epithelial cells are arranged in regular columns with nuclei at the base. The basement membrane is very thick and appears to form the endosteum of the cysts. The contents of the cysts are homogeneous in character. (Fig. 8.)

In the chronic nonsuppurative forms of sinusitis, where the symptoms of asthma are not present, the minute pathology appears to differ in one main point, in that the rarefying osteal change is not as prominent; and the writer wishes to call attention to this for the purpose of throwing some probable light on the etiology of the asthmatic symptom. The structure covering the bone has about the same type of change as in the asthma cases; that is, the absence of the round cell infiltration.

The inflammatory edema and secondary myxomatous change are very frequently found at the expense of degenerative changes of the mucous glands. In many of the sections one can make out the beginning of polypus formation. Of course, there are quite a number of sections encountered in which a mixture of inflammatory as well as noninflammatory changes is found, as will be shown in connection with the description of the suppurative type of chronic sinusitis.

In reference to hay fever, it has been argued by many that the sinuses are perhaps the seat of the pathologic change, and treatment directed to them has been reported of value, especially the removal of middle turbinates and curettage of the ethmoid, also washing out and treatment of the cavities, as the antrum, frontal and sphenoid. In operating on a number of these cases and examining the tissues histologically, I have found nothing markedly changed in the middle turbinates. The mucous glands seemed perhaps to be in the state of hypertrophy. (Fig. 5.) Of course, the operations were always done long after or preceding an attack.

In regard to the chronic sinusitis of the suppurative form, we find that the principal change in the early stages is that of marked round cell infiltration, especially in the subepithelial structure (Fig. 12), and the glands appear to be distended in areas, while, in others, destroyed by the inflammatory change. The epithelium is very frequently thickened and inspissated with pus (Fig. 12A), and the bone is seldom changed.

Later, however, the connective tissue fibrosis is found quite prominent, and in many instances the bone is also infiltrated with round cells. (Fig. 12B.) If the process has been of long standing, areas of necrosis in the bone will often be found, especially in curetted portions of the ethmoid. (Fig. 12C.) In connection with this necrotic process distinct areas of granulation tissue are encountered, and in some instances necrotic bone in the form of small sequestra.

In regard to the lining of the cavities, as the antrum, frontals and sphenoids, there is found in the long protracted cases a marked thickening with no evidence of any glandular structures, and many areas of myxomatous degeneration (Fig. 12D); again, a great deal of round cell infiltration and a thin layer of flattened epithelium. (Fig. 12E.) This latter condition is, however, not constant, for at times the epithelium is markedly thickened. (Fig. 12F.) The importance of the epithelium, however, is self-evident, in that its presence prevents spontaneous obliteration of the cavities, and its complete eradication is necessary in operative procedures in order to obtain this result.

In reference to the surgical pathology of polypi, we have found the most frequent condition to be a fibrosis with areas of myxomatous degeneration and inflammatory edema. The round cells are not very prominent except near the surface. (Fig. 12G.) The epithelium covering the polypi will depend on whether there is present pus or secondary recurrence after operative procedures. In the latter condition, the epithelium is markedly thickened (Fig. 12I); in massive polypi, which are crowded within the nares, also in the multiform polyposes, in which the nares are distended by polypi, the epithelium covering is thick (Fig. 12H). As to solitary polypi, especially those of the sphenoid, we have noted a cyst formation. (Fig. 12I.) These cysts are not at all uncommon in the polypi of the nonsuppurative form of sinusitis.

In assuming that atrophic rhinitis belongs to the group of

sinus involvement, either primary or secondary, we find the pathologic changes in the middle turbinate to be as follows:

In the early stages the bone has many areas of rarefaction. The soft structure shows areas of marked degeneration in the glands, and the epithelium covering the turbinate exhibits early tendencies to metaplasia or hornification. (Fig. 1.) Later, the bone shows a disposition to shrink, due to the preponderance of fibrous connective tissue. The glands practically are all destroyed, and the epithelium has still greater metaplastic change with many areas of denudation. (Fig. 10.) Similar changes are found in the curetted portions of the ethmoid in atrophic rhinitis.

CONCLUSIONS.

First.—The pathologic changes found in the middle turbinates and curetted portions of the ethmoid in asthma cases and in nonsuppurative sinusitis are very striking in that the bone shows rarefaction somewhat resembling that found in early bony changes of osteomalacia, acromegalia and otosclerosis. This pathologic finding is suggestive of a possible etiologic factor in some disturbances of the polyglandular system or the glands of internal secretion.

Second.—In the nonsuppurative sinusitis the conspicuous absence of inflammatory elements, as the round cell infiltration, and the presence of inflammatory edema or myxomatous degeneration at the expense of loss of glandular structures, is very apparent.

Third.—In the suppurative forms of sinusitis the great prevalence of round cell infiltration with tendencies to necrosis and granulation formation is very characteristic.

Fourth.—The lining of the larger sinuses, antrum, frontal and sphenoid, in the markedly protracted suppurative types, is so changed as to preclude resolution, and no matter how well these cavities may be drained or ventilated, their obliteration or eradication cannot be brought about unless the entire epithelial lining is destroyed.

Fifth.—The pathologic characteristics of both suppurative and nonsuppurative forms of sinusitis are very frequently met with in combination in the same case.

Sixth.—The great similarity in the changes of atrophic rhinitis in the early stages and of nonsuppurative sinusitis, especially in the bone, would suggest a similar etiologic factor in the disturbances of the glands of internal secretion, as mentioned in first conclusion.

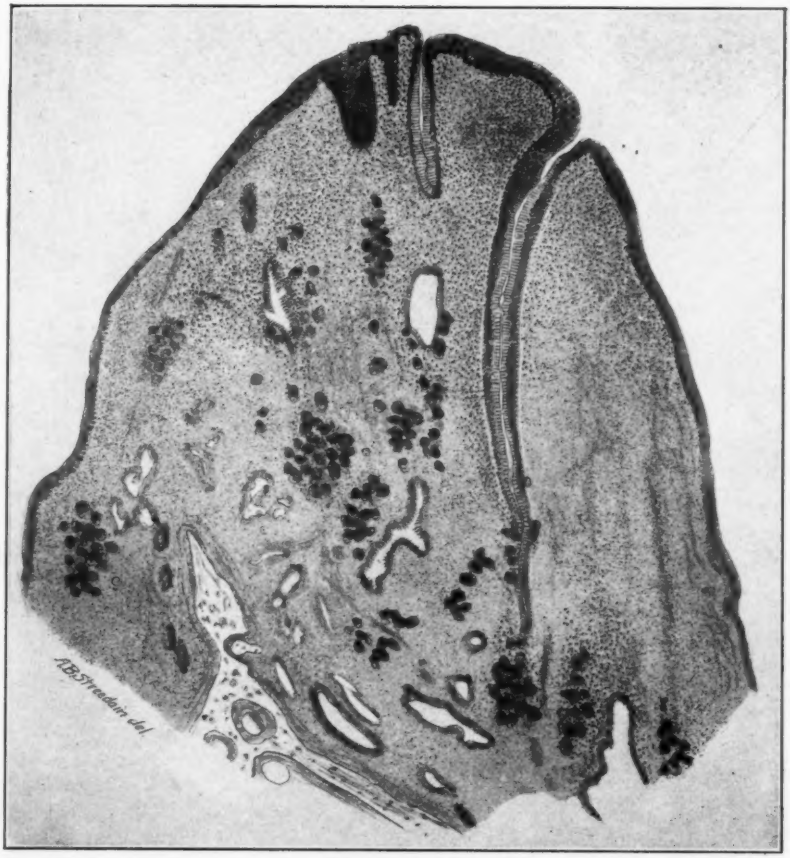

FIGURE 1.

Middle turbinate. Atrophic rhinitis.

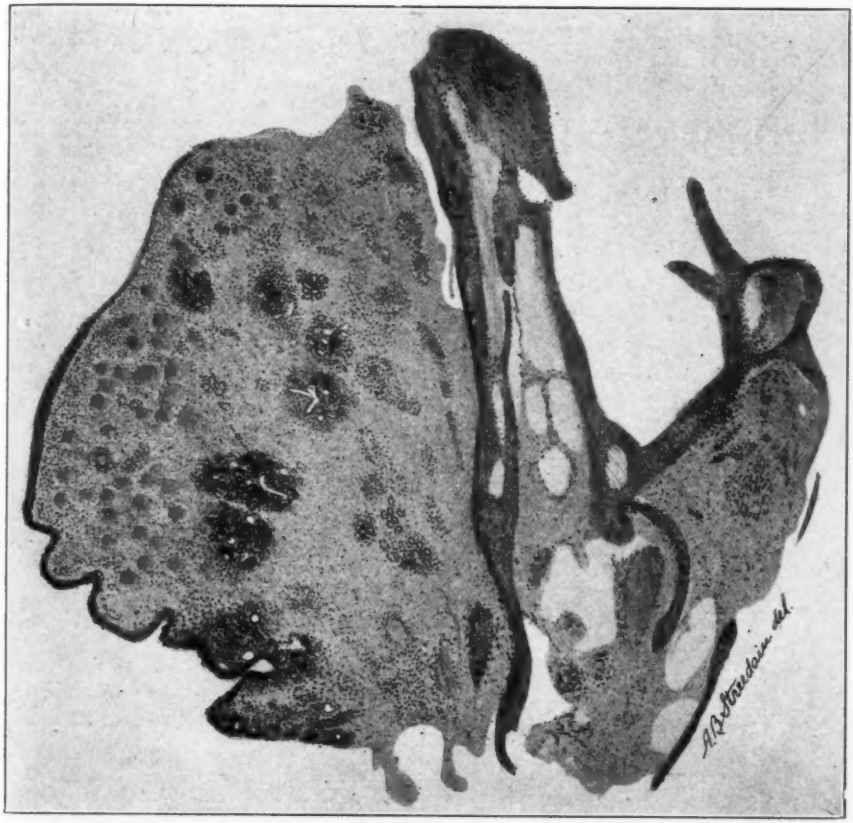

FIGURE 2.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

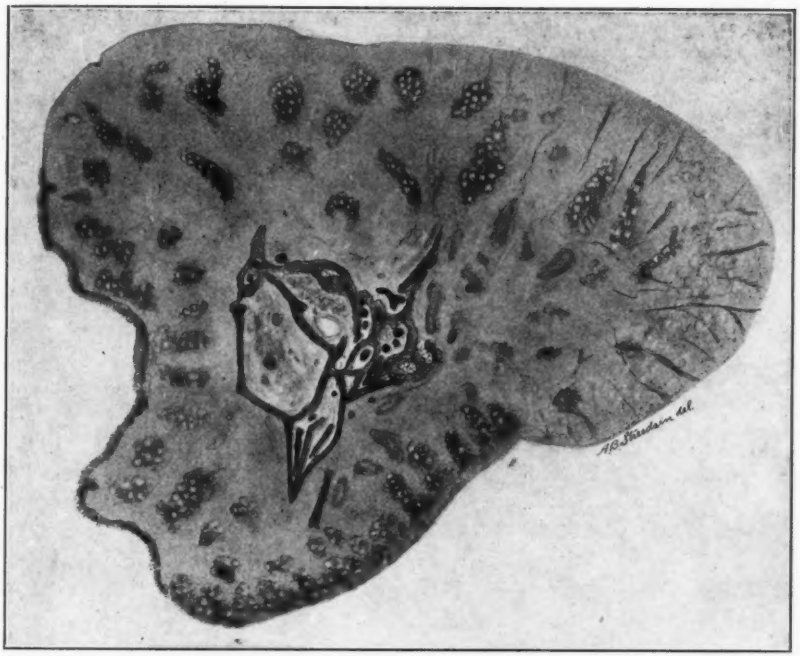

FIGURE 3.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

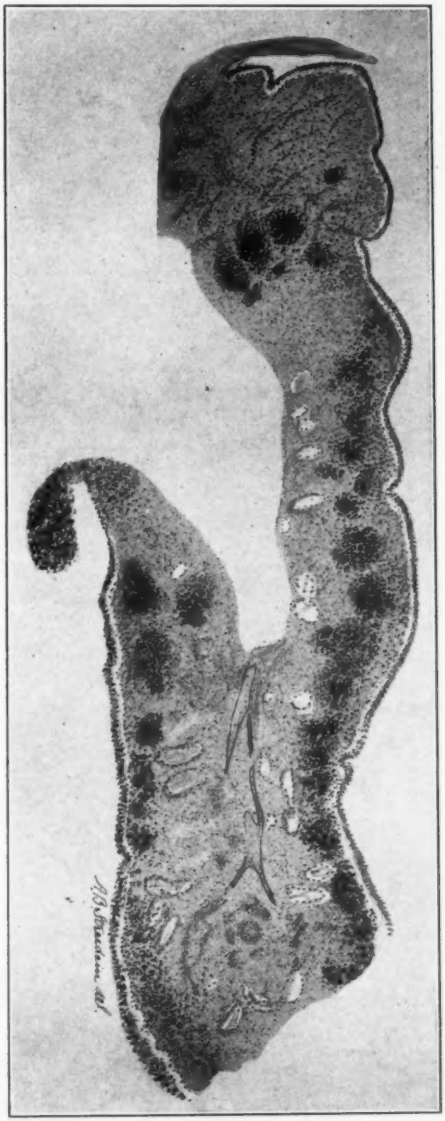

FIGURE 4.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

FIGURE 5.

Middle turbinate. Hay fever. (Asthma.)

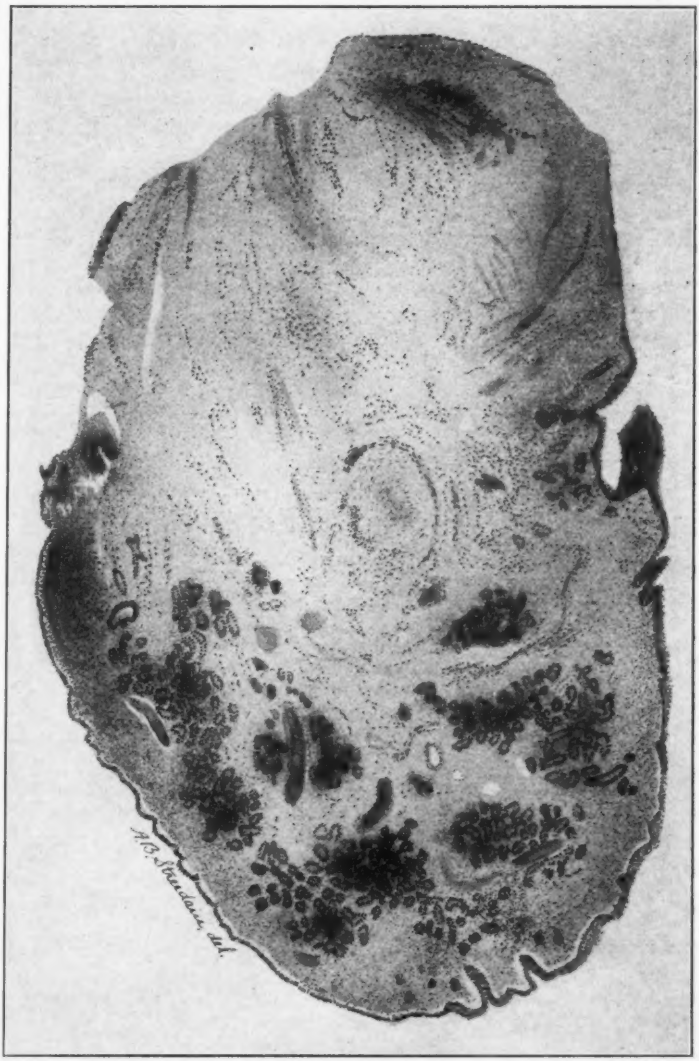

FIGURE 6.

Middle turbinate. Nonsuppurative sinusitis. (Asthma.)

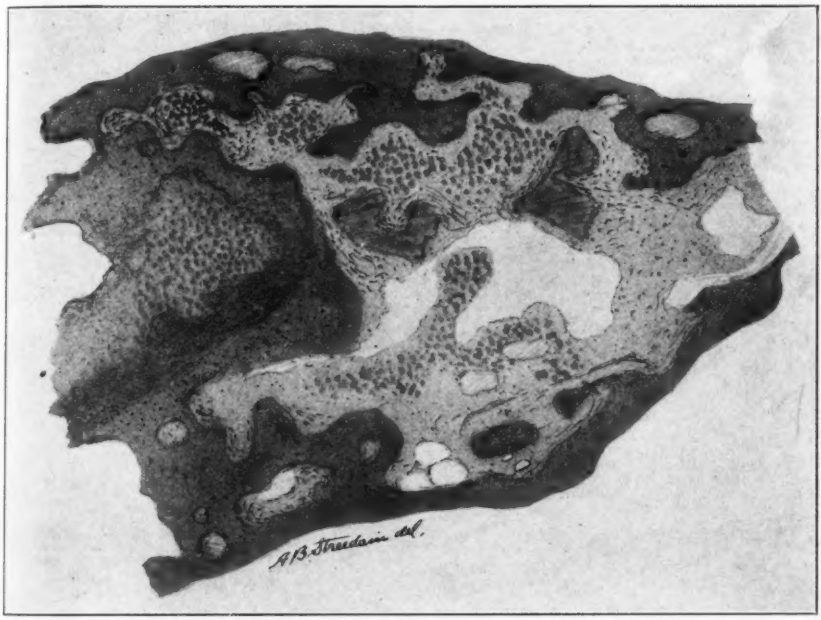

FIGURE 7.

Middle turbinate. Nonsuppurative. (Asthma.)

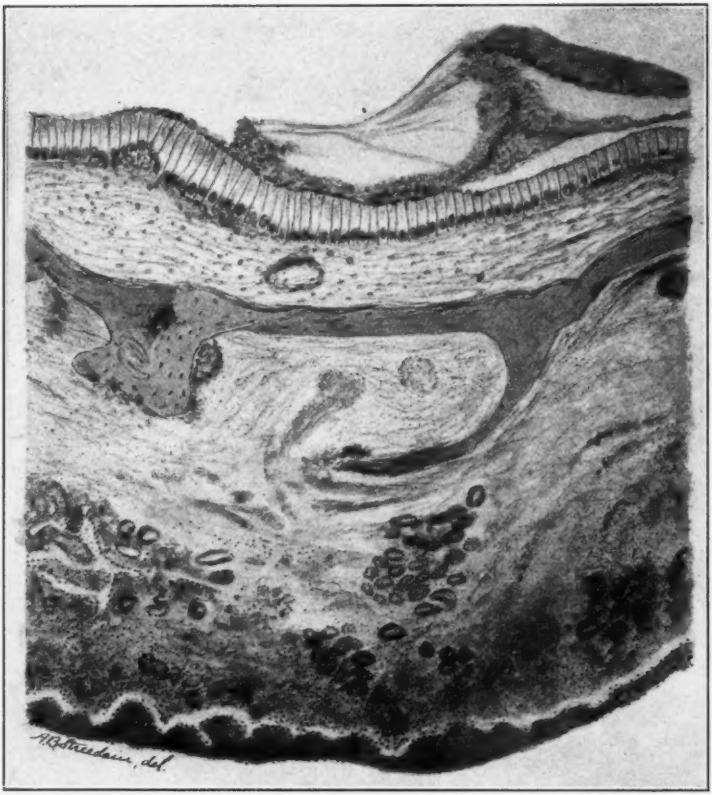

FIGURE 8.

Middle turbinate. Cystic formation. Nonsuppurative sinusitis. (Asthma.)

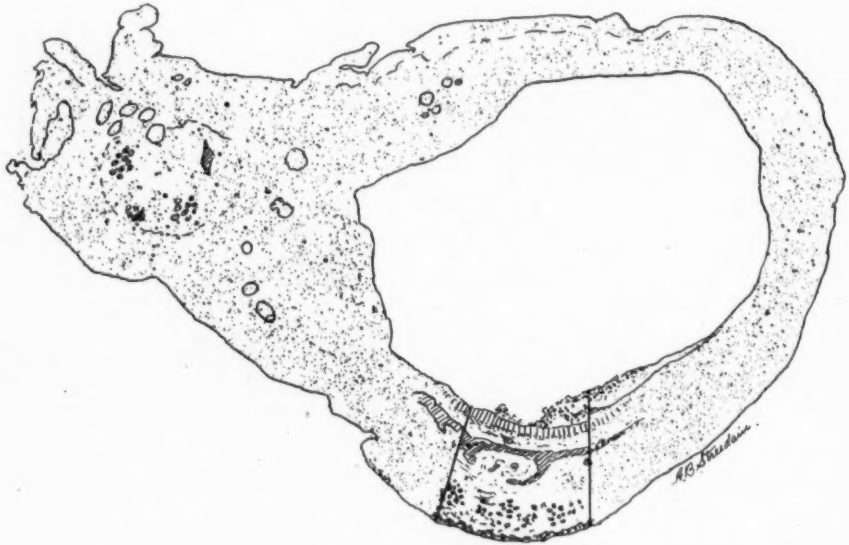

FIGURE 9.

Key to Figure 8.

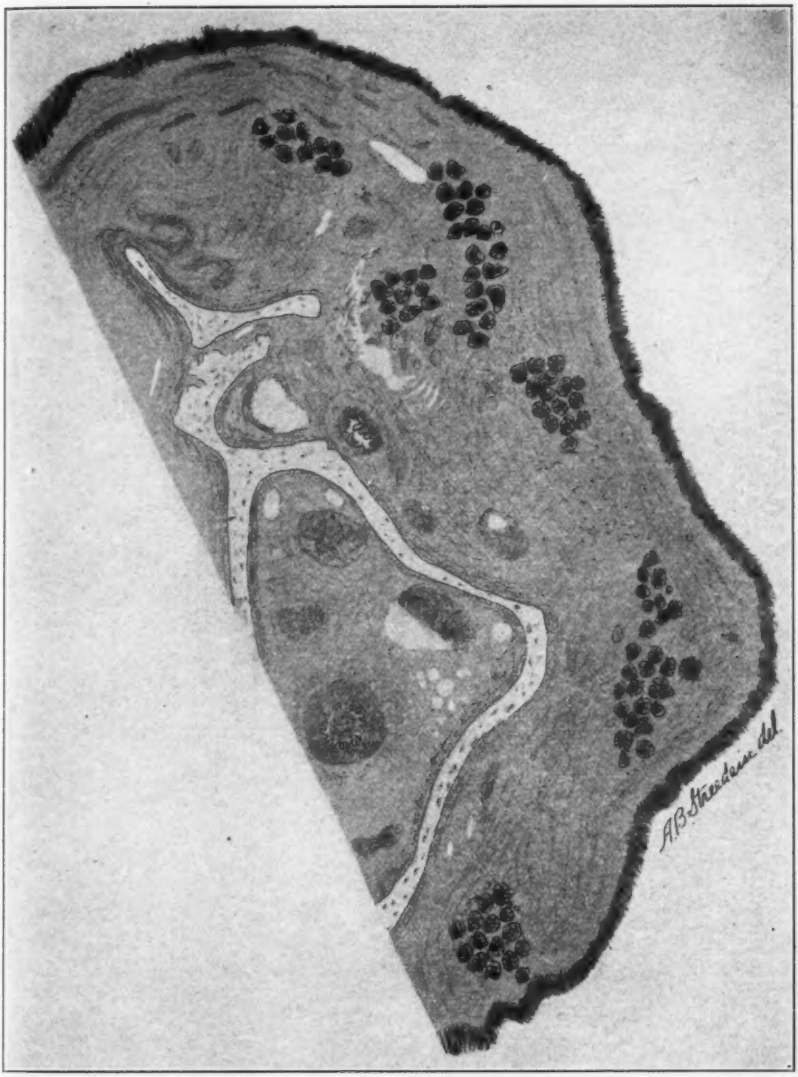

FIGURE 10.

Middle turbinate. Atrophic rhinitis. (Late stage.)

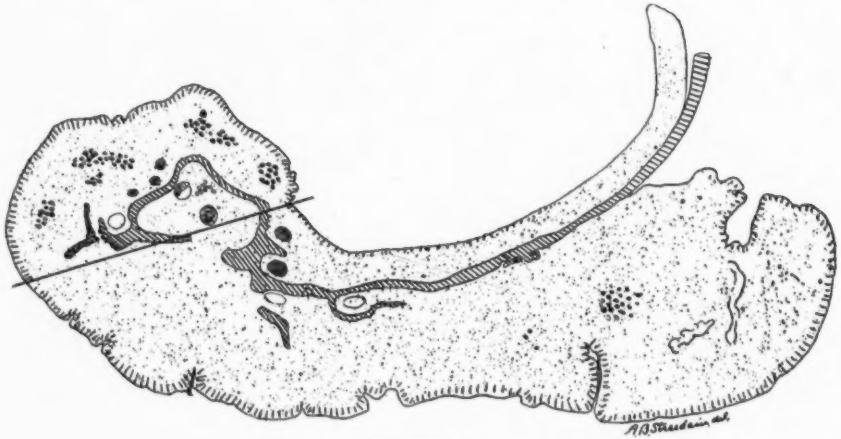

FIGURE 11.

Key to Figure 10.

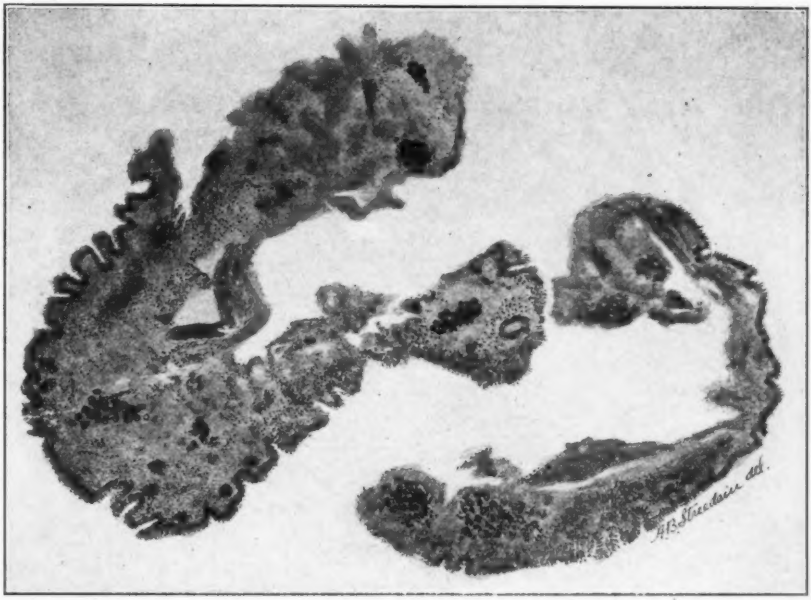

FIGURE 12.

Ethmoid cells. Chronic suppurative sinusitis. (Asthma.)

FIGURE 12A.

Necrosing ethmoiditis and chronic infiltrated hypertrophy of the mucous membrane.

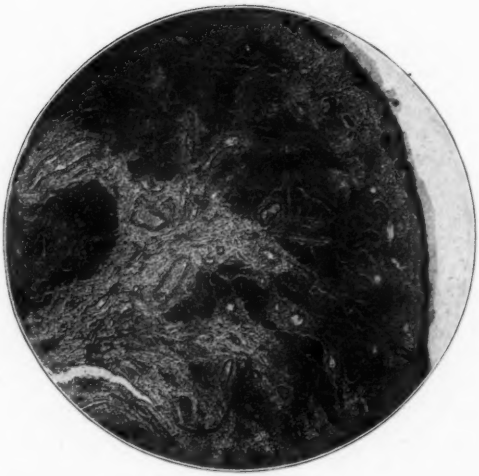

FIGURE 12B.

Chronic glandular hypertrophy of middle turbinate in chronic suppurative pansinusitis.

FIGURE 12C.

Rarifying ostitis of the ethmoid cell in nonsuppurative sinusitis.

FIGURE 12D.

Lining membrane of the frontal sinus in chronic suppurative sinusitis.

FIGURE 12E.

Pyogenic membrane lining the antrum of Highmore in chronic suppuration of all the sinuses.

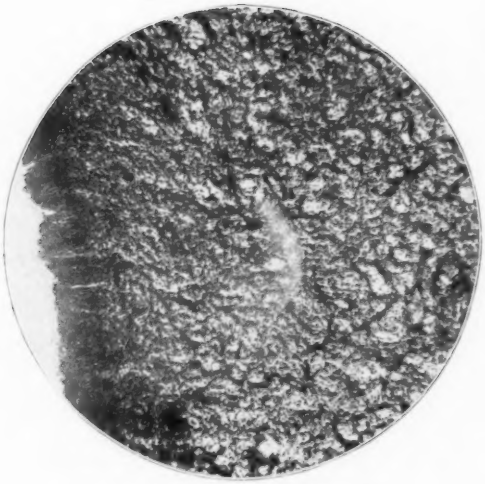

FIGURE 12F.

Solitary fibromyxomatous polypus of the frontal sinus in chronic suppurative pansinusitis.

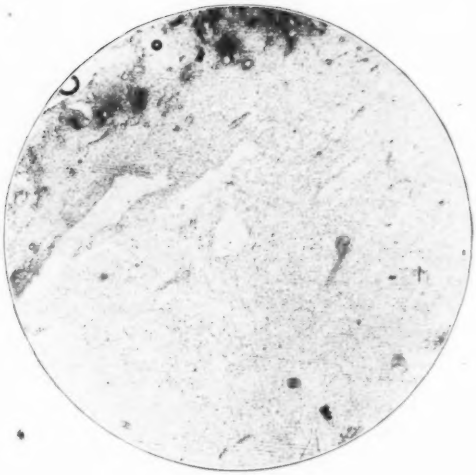

FIGURE 12G.

Inflammatory edema of a nasal polyp with cystic formation in chronic nonsuppurative sinusitis.

FIGURE 12H.

Inflammatory edema and hypertrophy of epithelium of a large nasal polypus in nonsuppurative sinusitis.

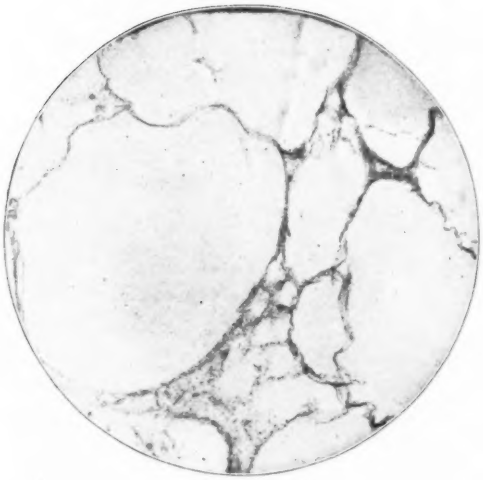

FIGURE 121.

Cystic formation in a nasal polyp in a nonsuppurative sinusitis.

FIGURE 13.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

FIGURE 14.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

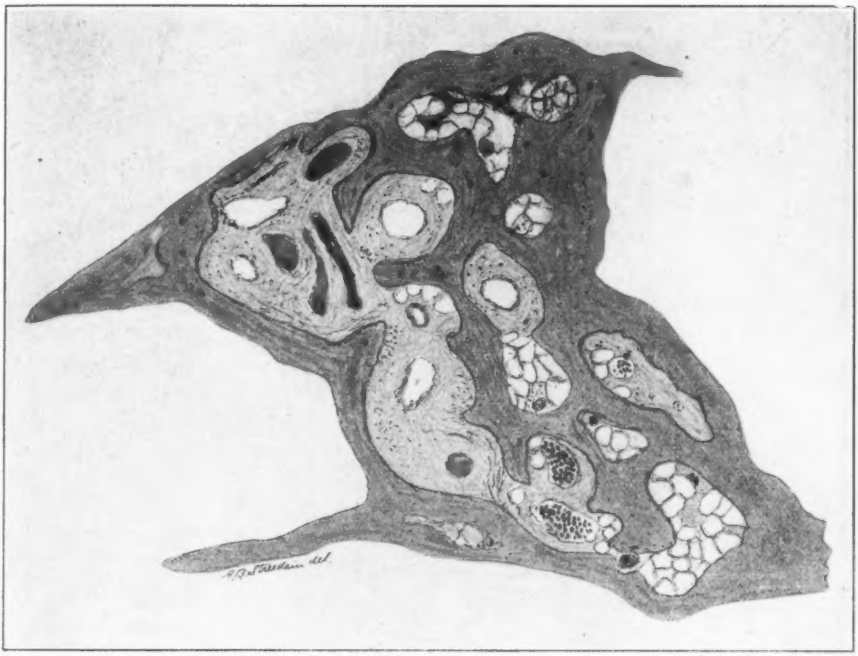

FIGURE 15.

Middle turbinate. Chronic nonsuppurative. (Asthma.)

FIGURE 16.

Middle turbinate. Chronic nonsuppurative sinusitis. (Asthma.)

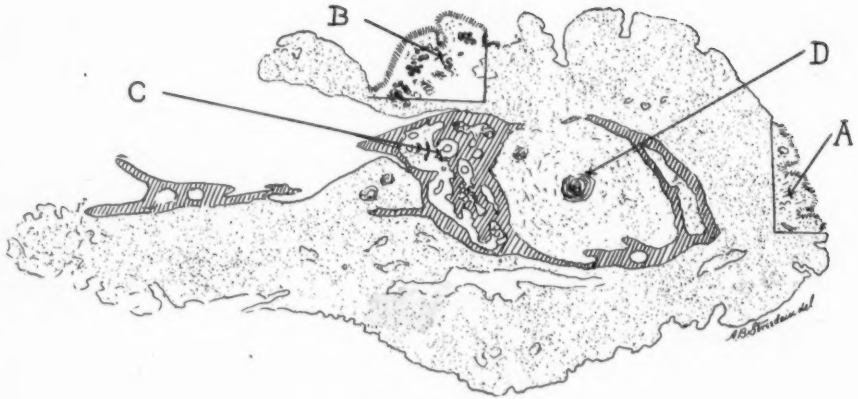

FIGURE 17.

Key to Figures 13, 14, 15 and 16.

LVII.

THE ENDONASAL ROUTE OF ATTACK IN HYPOPHYSEAL TUMOR CASES.

By W. E. Sauer, M. D.,

St. Louis.

As late as 1882 Hyrtl stated that the sphenoid cavities were entirely beyond the reach of hand or instrument. Today the modern rhinologist deals with these sinuses as a part of his regular routine, and even employs them as a route for entering the cranial cavity. After experimental research and clinical observation had proven that the disorders of the hypophysis were amenable to surgical intervention, the rhinologist was not long in proving that these diseases were within his province, in fact he has been able to show a lower mortality rate than others not working along this line.

The hypophysis has been reached by two different routes: the intracranial and the extracranial or transphenoidal. The intracranial route was first suggested by Canton and Paul, and later employed by Horsley. McArthur attempted to attack the hypophysis through the anterior cranial fossa in 1908, but failed. Bogoisolensky performed the first successful operation by this route in 1912. Schloffer, however, performed the first successful operation in 1907, employing the transphenoidal route. He found it necessary to remove practically the entire nasal interior, as well as temporarily to resect the external nose. This operation was later modified by Koenig, Knavel and Halsted, but to Hirsch of Vienna belongs the credit of developing the endonasal operation. He conceived the idea that the sella turcica could be opened through the nose without the preliminary operation of severing the external nose from its attachment and turning it to one side, as was done by Schloffer. The real point in Schlof-

fer's operation was the opening of the sella turcica through the sphenoid. To accomplish this he and others found it necessary, in order to have sufficient room to work, to resect first and then to push aside the external nose. The real difficulty, as stated by both Schloffer and von Eiselberg, was to keep their bearings after they entered the ethmoid. It became extremely difficult for them to tell if they were still within the ethmoid or had reached the sphenoid. Von Eiselberg attempted to take his measurements from a radiograph of the patient. In one instance at least it was necessary to call on the anatomist Tandler to assist him in finding the sphenoid cavity.

Hirsch carried on some experiments on the cadaver, and attempted to open the sella without disturbing the nasal structures other than removing the middle turbinate. He succeeded in making an opening in the floor of the sella 1 cm. by 5 mm. wide, without injuring any of the surrounding structures. These experiments were later controlled by Dr. Erdheim of the pathologic institute of Vienna. Hirsch had also observed in all of his work on the sphenoid sinuses on the living, how much of the upper wall of the sinus could be seen and how relatively easy it would be, especially when there was a large sinus, to open the wall and if necessary to remove a part of it. His studies of a large number of radiographs of the skull still further confirmed his views.

Hirsch developed two methods of attack through the nose:

First, the ethmoidal route. This consists of the removal of the middle turbinate with the exenteration of the ethmoid labyrinth on the same side. This gives a free exposure of the anterior wall of the sphenoid. Its anterior wall is then removed as far as possible, and the upper wall or floor of the sella turcica is opened with a chisel, and this opening is enlarged with a suitable forceps.

The second is the septal route. This consists of a complete submucous resection in the usual way. In addition, the mucous membrane covering the anterior wall of the sphenoid is pushed to each side until the openings of the sinuses are visible. The entire bony septum having been removed, the openings of the sphenoid are enlarged and the partition between the two cavities is then removed. This gives a free access to the upper

wall of the sphenoid, which is opened, and the opening enlarged as before. In this operation the work is done entirely between the two mucous membranes of the septum. If the middle turbinate must be removed, it should be done several weeks before. The advantage claimed for this route is that all the manipulations are carried on within a sterile cavity without entering the cavity of the nose. This, however, is not alwaays possible, because the mucous membrane is not so easily deflected from the anterior walls of the sphenoid, and as the mucous membrane is rather thin at this point, it is easily torn and the cavity of the nose is entered; at least this has occurred in my case.

About the same time that Hirsch was doing his work, West of Baltimore brought forth a method which he worked out on the cadaver. His method consists of a partial removal of both inferior and middle turbinates. Enough of the turbinate is removed to give a good view of the anterior wall of the sphenoid. Without elevating the mucous membrane of the septum, he makes a vertical incision through the cartilaginous septum, and then by means of a chisel makes a cut, beginning at the upper end of the first incision and extending to the upper part of the posterior end of the septum. A second is made from one-half to three-fourths inch below and parallel to the upper cut. The bone is then removed; this gives a free access to the sphenoid and gives a large space to work in. The anterior wall of the sphenoid is then removed with a chisel. The septum of the sphenoid is next removed and the sella opened, as in the Hirsch operation.

Cushing makes an incision under the lip, and then does a complete resection of the septum through this incision. He claims that in starting under the lip there is about twice as much room than that secured by the Hirsch method. Cushing calls attention to the difficulty in avoiding a tear in the mucous membrane in elevating it from the cartilaginous septum. He also points out the possibility of entering the ethmoid and mistaking it for the sphenoid. In one case the cribriform plate was opened, owing to the fact that the angle of attack was directed too high. This error is not likely to occur with a patient under cocain anesthesia, if the Hirsch or West technic is followed.

The chief objection to the intranasal route has been the dan-

ger of infection, resulting in meningitis. It has been argued that the nose is not a sterile cavity, and that it is impossible to make it sterile. This, however, has not been borne out by experience. Hirsch has reported twenty-six cases of hypohyseal tumor which he has operated upon, with only three deaths. One death was due to a pneumonia; in another, a hemorrhage occurred into the third ventricle; and in the third, a meningitis. In this case the patient was a dement, who removed the packing himself. The results of Hirsch's operations are better than any series of operations reported by any other writer. Cushing had four deaths in twenty-nine cases. Von Eiselberg had four deaths out of fourteen cases. In thirty-two operations according to Knavel's method, six deaths occurred.

Spiess and Holmgren each report a case operated upon by the Hirsch method, with good results. Adding my own case to this makes a total of twenty-nine cases, with a mortality of 10.3 per cent as compared to Cushing's 13.7 per cent. Furthermore, Hirsch operated on one of his cases six times and another four times. These secondary operations should be considered in making up the percentage, as these operations exposed the patient to the same danger as the primary operations; when these are added it brings the mortality rate down to 8.1 per cent. These figures would prove that the danger of infection is more apparent than real. Schloffer has further shown that the hypophysis differs from other portions of the brain, in that while all the other portions of the brain are covered by the pia, arachnoid and dura, the sella portion of the hypophysis is covered only by the dura. The subarachnoid space is not opened when only the sella portion of the tumor is removed, thereby greatly lessening the danger of infection.

Cushing states that as a matter of fact the risk of inducing an infectious meningitis is comparatively slight when the turbinates are not removed and the mucous membrane is not lacerated. Even the existing risk is greatly lessened by the application of Crow's discovery, that hexamethylenamin finds its way into the cerebrospinal spaces. This drug is given in doses up to sixty grains in twenty-four hours, by mouth, on the day preceding and for some days after the operation. Von Eiselberg reports two deaths from an infectious meningitis following his modification of the Schloffer operation. He attributes this in-

fection to an existing acute rhinitis which had been overlooked or underestimated by him. One case died within thirty hours and the other four days after the operation.

In looking over the literature I find reports of thirty cases operated upon by the endonasal route; twenty-six of these were performed by Hirsch of Vienna, one by Spiess, one by Holmgren, one by Pierce, and one by Morshik. In summing up his results Hirsch states that three deaths occurred: one from pneumonia, one from hemorrhage into the third ventricle, and one from meningitis. In three cases there was no improvement following the operation; in two of these cases there was a marked defect in vision of long standing; the other case was an old case of acromegaly. He concluded that in these cases the principal parts of the tumor had grown upward, and the small parts removed had no influence on the symptoms, or else the optic nerves had been destroyed. In five of the cases the relief obtained was only of short duration—two to six months. In sixteen cases the results were good and the improvement obtained has been maintained. In this group are a number of cases in which there has been a marked improvement in vision as well as improvement in the general symptoms. In one case two years have elapsed since the operation with no recurrence of the symptoms.

Pierce of Chicago reports a case of increasing blindness, diplopia and headache, in which a growth in the sphenoid sinuses was suspected. The middle turbinate was removed and the ethmoid cells were opened. The anterior wall of the sphenoid wall was then removed and a slight amount of mucopus was evacuated. The lining membrane of the cavity was pale and smooth. The striking feature was the small anteroposterior diameter of the cavity. When touched with a probe it conveyed a sensation of bogginess; marked improvement followed this decompression. The case was later operated on by Dr. Halsted, by a modified Schloffer.

Spiess reports a case in a patient twenty-eight years of age, in which the diagnosis had been confirmed by the X-ray. He followed the technic of Hirsch, and found, instead of the posterior wall of the sphenoid cavity, a tumor about the size of a hazel nut, which proved to be a cyst. In attempting to remove this cyst it was ruptured. The contents were removed and as

much of the wall as possible was curetted away. The patient ran a temperature for a time, but made a good recovery. Vision was restored, with marked improvement in the general symptoms.

Holmgren reports a case of a man aged forty, who complained of headache with marked impairment of vision. He had a bitemporal hemianopsia, contracted visual field. X-ray revealed a destruction of the bone beneath the sella turcica. Both middle turbinates were removed. A submucous resection of the septum was done. The anterior wall of the sphenoid was removed. The tumor had destroyed a part of the posterior wall of the sphenoid. No attempt was made to remove the tumor. Marked improvement in vision and general symptoms followed.

The case operated upon by Morshik was a patient of von Eiselberg's, in which the latter had done a temporary resection of the nose; he opened the ethmoid and a quantity of mucopus was found. A pulsating tumor was seen in the region of the sphenoid, but owing to the unexpected presence of pus in the ethmoid, von Eiselberg decided not to attack the tumor; the wound was packed with gauze, the nose was stitched back in place and the gauze tampon removed on the seventh day. Dr. Morshik was called in to see the case; he found large quantities of pus in the epipharynx. Four weeks later the patient developed an acute angina, which necessitated the postponement of the operation. The mucopurulent discharge finally ceased, and two months after the first operation Morshik attempted the endonasal removal of the tumor. The patient was given a morphin scopolamin injection one hour before the operation, and the mucous membrane infiltrated with a 1 per cent novocain solution. This tumor was found to be covered with granulations which had to be removed. The opening in the sphenoid was enlarged. On attempting to remove the tumor it fell to pieces on the floor of the nose. A free hemorrhage followed; this was controlled by packing with gauze; the gauze was removed after a few moments and a probe introduced. The probe passed into the cranium for quite a distance without any resistance. The gauze was again gently packed into the wound and removed one hour later. The tumor proved to be a sarcoma. The evening after the operation the patient had a

high temperature with headache. Patient died in forty-eight hours, from meningitis.

HYPOPHYSEAL TUMOR WITH ACROMEGALY.

In conclusion I wish to report the following case referred by Dr. Behrens, to whom I am also indebted for the history and the physical findings:

S. D., aged thirty-two years, white; coal miner formerly; saloon keeper now. Lithuanian (Russian Poland).

Complaint.—Was sent to me in October, 1911, because of palpitation of and oppression over heart, causing anxiety and fear; weak generally; growing weaker. Also had gastrointestinal disturbances without undue provocation.

Family history.—Father died at sixty-seven years of age, cause unknown; was of normal proportions. Mother living, age seventy-five years, and well; also of normal physique. Brother living and well, age forty-two years; large, over average height and weight. Sister died at fifty-two years, cause unknown. Family were all large, but not ill proportioned. Knows of no nervous, cancerous, tuberculosis or kidney diseases in family.

Previous history.—General health splendid until seventeenth year of age. He has had the milder exanthemata of childhood, but no scarlet fever, diphtheria, rheumatism, chorea, typhoid fever, pneumonia or malaria. He denies venereal disease or sexual overindulgence, and has been moderate in habits as to tobacco or drink.

Personal history.—At the age of seventeen he began to have slight headaches, never severe, but at times periodical and constant, frontal in character and especially early mornings. Some ten years ago the symptoms increased, with visual disturbances added, most prominent in left eye; then gradually the right eye began to show similar disturbances, but he cannot see as well now with the left eye. He suffers from cardiac excitability.

Examination.—Head—No deafness, no ear discharge, odd feelings in head.

Cardiorespiratory.—No cough, no dyspnea, no edema; perspires easily.

Gastrointestinal.—Good appetite, some dull pains in stom-

ach and around heart, no nausea, no vomiting, eructation at times of sour material; bowels regular.

Genitourinary.—Urinates regularly, no gonorrhea or syphilis.

Neuromuscular.—No convulsions, numbness or peripheral pains; has normal sexual power and desires. Quit work as a miner because of weakness and cardiac distress.

Examination.—Patient large, thick set, white male adult.

Abdomen.—Negative; no palpable enlargements of spleen or liver. Penis shows no scars, of good development; testes likewise of normal consistency. Normal knee jerk, no Babinsky, no clonus, no edema of legs or ankles; some veins enlarged. Both feet large, large toes, quite abnormally thick, others above normal, bones of legs and thighs not irregular or equally or portionately enlarged. Spade-like hands, large fingers, short thick thumb 9 cm., middle finger 9 cm. Teeth show marked separation owing to enlargement of lower jaw. Tongue is also large.

Ophthalmic examination.—Pupils not dilated, respond to light, discs unusually bright and shining, optic atrophy slight, no vessel engorgement, not tortuous. Vision good, right eye 15/12, left 15/200.

During year of observation very little difference in any condition, mental or physical, was noted, with the exception of marked decrease in vision in left eye.

Nose.—No discharge.

Over skin of thorax, abdomen and back are numerous nodules, firm, fibrous-like in feel, movable, not tender.

The indication for operation was the increasing loss of vision in the left eye. The patient was sent to the Deaconess Hospital on November 28th, and on the following day, after the usual preparations for a submucous resection of the septum, he was sent to the operating room. The septum and the region of the anterior wall of the sphenoid were carefully cocainized with a 20 per cent solution of cocain. In addition, a one-half per cent solution of cocain was injected on each side of the septum near its posterior end. The septum was deviated to the left anteriorly, and the right posteriorly. The middle turbinate was removed on the left side, after which a submucous resection was done in the usual way. The mucous

membrane on the anterior wall of the sphenoid was pushed to the side until the openings of the sphenoid were reached. These openings were enlarged and the partition between the two sphenoid cavities was then removed. The upper wall of the sphenoid was found to be much lower than usual; it appeared to be bulging down into the cavity. This wall of the sphenoid cavity was then opened by means of a chisel, and the opening enlarged by means of a punch forceps. The bone was not as thin as I had expected to find it, but there was no difficulty in removing it. An opening about 1 cm. by ¾ cm. was made. The dura was incised by means of a sickle-shaped knife. This incision was very painful, but I managed to make a crucial incision; the patient requested that I discontinue.

The mucous membrane covering the anterior wall of the sphenoid was torn to such an extent that I packed the nose in the usual way, and did not pack between the mucous membrane, as recommended by Hirsch. The pack was removed at the end of twenty-four hours; there was no unusual bleeding or any other usual symptoms following the operation. The patient noticed a marked difference in the heart's action, and stated that he felt much lighter; he particularly noticed that the foul odor he had previously noticed had disappeared. The patient left the hospital ten days later.

An ocular examination by Dr. Parker shows the following: Conjunctiva, cornea, anterior chamber and iris, normal. Reaction to light better in right than left. Accommodation better in right than left. Visual acuity in right normal, left 17/120; could not be improved.

Ophthalmoscopic examination.—Right showed very slight beginning atrophy; left fundus on the temporal side looked clouded and markedly thinned, and in places disappearing entirely. Nasal half vessels much more easily defined.

Color field.—Much contracted, especially temporal side of left eye, also interlacing.

December 10, 1912.—Vision, right normal, while left was much improved; vision 17/60.

January 2, 1913.—Patient feeling and looking much brighter; right eye, vision normal; left 17/50.

February 1, 1913.—Right normal, left still improving; vision 17/40.

Eleven months have elapsed since the operation, and his vision in the right eye is normal, while the vision in the left eye remains at 17/40. The patient has been working as a day laborer, unloading ice, etc., without any discomfort, whereas before the operation he could scarcely walk a block without having to stop to rest. He now wears a smaller hat as well as smaller shoes. He is also able to bring his upper and lower incisors together, while before the operation the lower teeth protruded about a half inch. Should there be a recurrence of the symptoms, I will attempt to remove a part of the tumor.

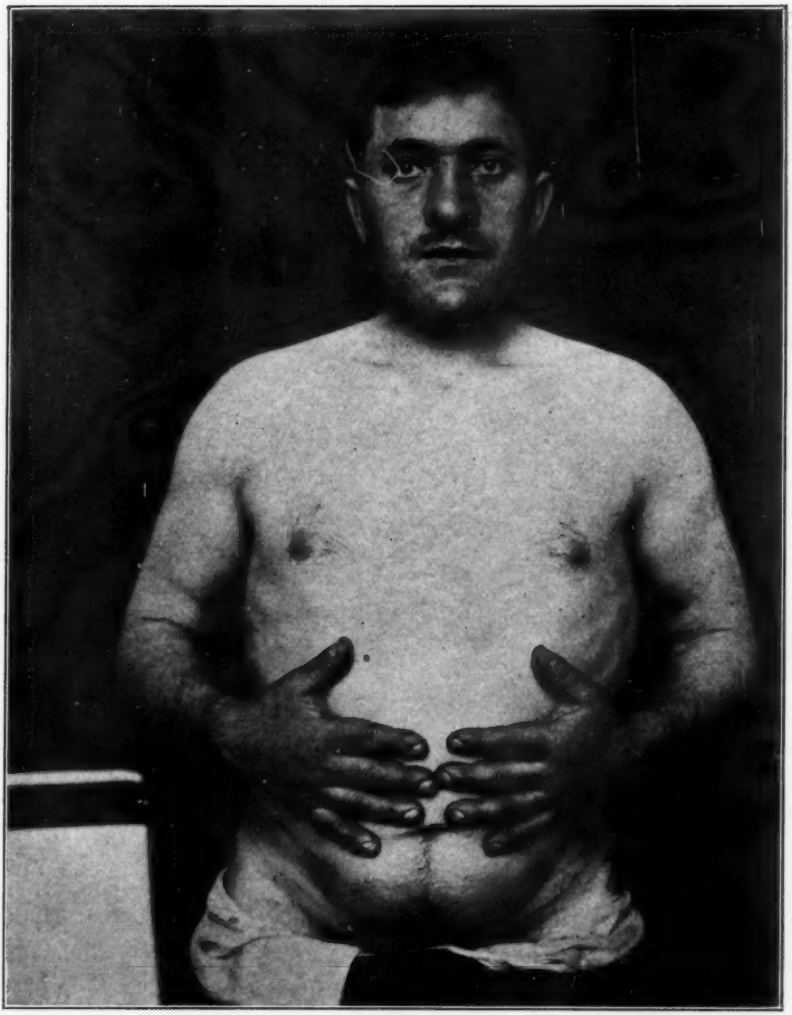

FIGURE 1.

Showing relative size of hands and body.

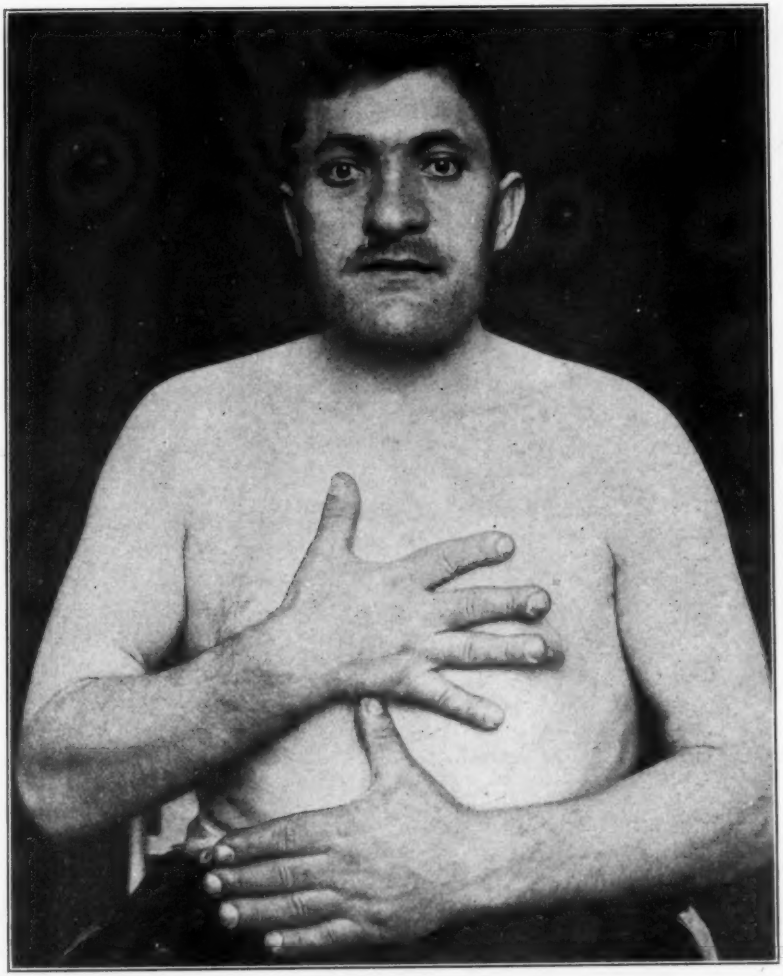

FIGURE 2.

Showing typical hands, chin and facial expression.

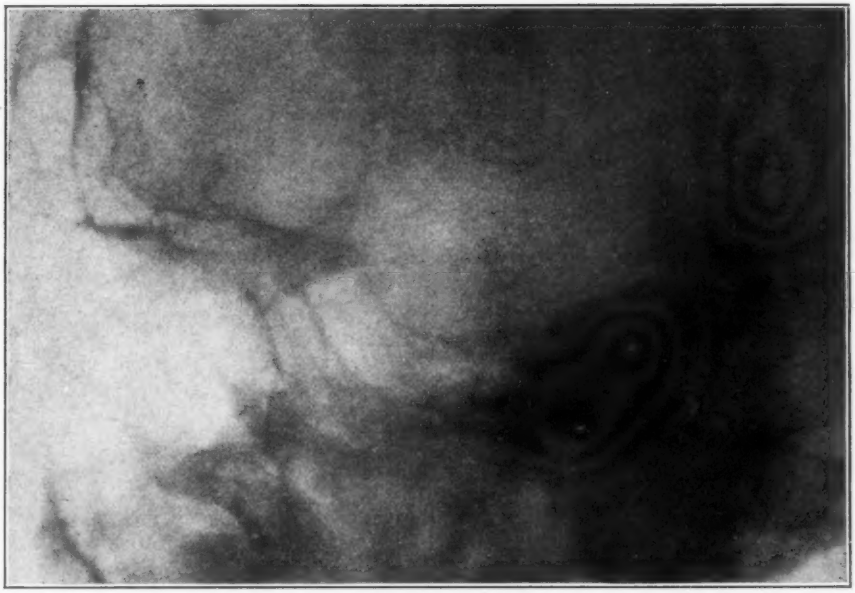

FIGURE 3.

Radiograph showing large sella turcica.

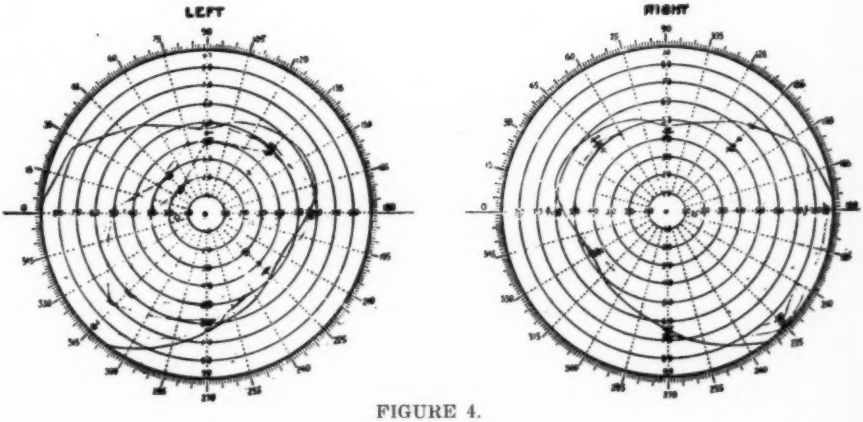

FIGURE 4.

Visual fields.

LVIII.

CAVERNOUS SINUS THROMBOSIS DUE TO ORGANISMS OF THE STREPTOTHRIX GROUP.

By Henry Horn, M. D.,

San Francisco.

Since 1907, when Löhlein reported his case of brain abscess resulting from a streptothrix infection, nothing of importance has appeared in the literature concerning this phase of the disease.

It is my good fortune to be able to add a little to the knowledge of this interesting subject and to report a case which seems to be absolutely unique in the scattered literature of the streptothrix infections.

Although there are numerous records of cases where the cause of death was proved to be an infection from streptothrix in various parts of the body, the cases combined with intracranial complications are comparatively rare, and, what interests us from the standpoint of otologists, intracranial complications originating from ear infections seem to be absolutely unknown.

In this article our interest has naturally been centered only on those cases in which the brain and its meninges have been involved.

The first case of this nature to be fully described and worked out bacteriologically was reported by Eppinger in 1890.[37] He was able to cultivate a mould from a metastatic abscess in the brain, and named the organism cladothrix asteroides, but this has since been identified as a true streptothrix and is now called streptothrix Eppengeri. He found this organism pathogenic for rabbits and guinea pigs, and was also the first to see the very great resemblance to tubercular processes and therefore designated the disease as pseudotuberculosis cladothricia, a

similarity which Flexner studied later and agreed to. In fact it is through this research that the recent work on the true botanical place of the tubercle bacillus was started.

In the same year Armquist also isolated a streptothrix from a meningeal exudate which occurred in connection with other mycelial forms.

Various fatal cases of streptothrix infections were now reported. Tschierschke (1891), Hesse (1892), Carten, Gruber (1893), and finally Riviere and Sabarez, in 1894, reported two cases, one of which again showed a metastatic brain abscess originating in a probable lung abscess. The streptothrix was isolated from both abscesses. In the same year Ferre and Faquet also isolated the organism from a brain abscess. Many interesting cases, not of intracranial origin, were reported by various authors, and again, in 1899, Ferrari described a metastatic brain abscess of this character, probably originating in broken down bronchial glands.

The next case which interests us was that of Chiari, who reported two cases of bronchiectasia which were complicated with meningitis. In 1903 both Horst and Löhlein reported a case in which multiple abscesses due to a streptothrix infection were found at autopsy, and again, in 1907, Löhlein had the good fortune to be able to report still another case of brain abscess of the same origin. From this time up to the present writing there seems to have been no other cases reported, and it can, therefore, be seen that the number of cases of brain complications following streptothrix infections is extremely small, probably not over ten all told; and as far as a careful review of the literature shows, none at all that started in the middle ear and later led to an involvement of the great blood sinuses of the brain.

The morphologic position of the streptothrix group is to some extent an unsettled problem, but it seems as if we ought to be willing to accept the carefully worked out life history, as given by Foulerton, who does not consider that the group should be classed as bacteria but as moulds.

If we look over the literature of the ear moulds, we find that the majority of the cases reported have usually been classed under the heading of mycosis, aspergillus or allied forms. These are clinically and morphologically unrelated to the streptothrix group, and as far as is known have never led to

intracranial complications. They are generally confined to the external auditory canal, but in a case reported by Haug, he described the mould as extending into the cells of the mastoid process, and only a few cases of middle ear infection have been traced to this source.

Like many other botanical groups, the nomenclature in the earlier works on this subject has been very loosely applied, and, therefore, there is considerable confusion as to whether the streptothrix is related to the actinimycoses. This, however, is not the case, for in the life history of the streptothrix there are such marked differences that their identity with the latter group is at once excluded.

As nearly as can be determined, the name streptothrix was first utilized by Corda in 1834, but the identify of his organism is somewhat in doubt. Probably the mould isolated by Cohn (1874) from the lacrimal canal is the same as we today recognize under the title of streptothrix. The further historical development of the subject has been worked out in Foulerton's article and need not be repeated here.

Inasmuch as it is the opinion of the author that this organism is far more common in otologic infections than has generally been reported, a short description of the morphologic characteristics will not be out of place. The description is borrowed freely from Foulerton's article and is here added, as it would seem practical to have the first article dealing with the subject from the otologic standpoint as complete as possible, and thereby obviate the necessity of reference to the original sources.

Starting from the original spherical spore, growing in artificial media, under suitable conditions of warmth and moisture, two or three small buds project and, gradually elongating, become the primary filaments of the mycelial mass. From the primary filaments lateral branches bud out, and from these secondary branches other lateral filaments again grow out by a repetition of the budding process, and in this manner a more or less dense and tangled tuft of wavy branching mycelium is formed.

The termination of the filaments presents various appearances in different species, and the appearance may not be constant in the same species; the end of the filaments may be pointed or rounded off, or may present small spherical or Indian-

club shaped enlargements. The adult mycelium presents a homogeneous appearance when stained with the ordinary basic anilin dyes or by Gram's method; when segmentation occurs there is an alteration of the staining properties, resulting from a degeneration of shorter or longer segments of the constituent filaments occurring at irregular intervals along their length. The degenerated portions of the filaments no longer retain the stain when treated by Gram's method, and stain but faintly with basic anilin dyes employed in the usual way. Fragmentation of the mycelium follows on segmentation, the degenerated segments gradually disappear, and in the place of the original tuft of branching mycelium there are collections of separate, shorter or longer, lengths of the filaments, which may appear as straight or slightly curved rods, or may retain the wavy outline of the original mycelium, or may present a well-marked corkscrew or spirillum shape.

It is quite certain that the rod segments never give rise directly to new rod segments by a process which is in any way analogous to the fission process which is a characteristic of the bacillary forms of the schizomycetes.

From the above description and from observation it can be stated that the morphology of the streptothrix is widely different at different stages.

In some of the pathogenetic species of streptotricheæ it is impossible to trace the complete morphology on any sort of media, the mycelium develops very slowly, and segmentation and fragmentation of the filaments appear to occur coincidently with the growth. In such cases the ordinary cover glass preparation will show little more than closely packed masses of rod segments which often have a beaded appearance, the whole closely resembling what is seen in a similar preparation from a culture of bacillus diphtheriæ or bacillus tuberculosis.

With but a few dissenting voices, the place which the genus streptothrix belongs in botanical classification is among the hyphomycetes or mould fungi. The dissenters from this view would regard the branching mycelium, the rod segments, and the spherical spores as merely casual morphologic variations of a highly pleomorphic fission fungus, which under different conditions may assume a branching filamentous, a spirillar, a bacillary, or coccal form.

The streptotricheæ, both the saprophytic and the pathogenetic forms, are very widely distributed in nature. In the higher animals we have the streptothrix bovis communis and streptothrix nocardii, occurring commonly in cattle. Numerous other species have been described, but as yet no carefully worked out case of these species as occurring in man has been found.

One or two fairly dependable cases of streptothrix bovis communis have been reported as occurring in man, but other than these few cases which might point to the lower animals as a source of infection, we are at loss to give a satisfactory explanation of the original source of the infection.

The frequency of primary streptothrix infection of the mouth in cattle, resulting in the formation of small nodules situated immediately under, and sometimes actually invading, the mucous surface of the dorsum of the tongue, has obviously suggested that herbage may be a special habitat of the streptotricheæ and a possible source of infection. Further, the general belief that these infections are especially liable to occur among those whose occupations bring them into frequent contact with dust emanating from dried grass, is to some extent supported by a consideration of the habits and occupation of thirty-nine individuals above mentioned. Thus, there were four men employed in stables, constantly handling hay and straw, and possibly addicted to the habit, prevalent among those employed in stables, of chewing pieces of straw. Two of the patients were gardeners, two cases occurred in bakers, and one patient was a dairy maid.

There are certain new features covering the pathogenetic action of this organism in the case about to be described which make it necessary to refer once more to the general literature and to show what the generally accepted opinion is regarding this point.

The only cases which are at all similar to ours are the ones which refer to intracranial complications. Here the occurrence of the metastatic brain abscess is the predominating type. As seen in other parts of the body, the infection causes the development of nodules and larger masses of granulomatous tissue around the invading parasites. These masses may cicatrize or break down into abscesses.

Speaking generally, it may be said that the streptotricheæ

which are the cause of human infection are not of themselves apt to produce any very marked constitutional symptoms or effects, either by the secretion of actively virulent toxins or by the production of like bodies by decomposition of the tissue in which they may be growing.

The uncomplicated action of the streptotricheæ, then, is not usually manifested by severe general symptoms, but rather in the physical results of the tissues actually infected. Generally it is only when the granulomatous tissue, having broken down, has become the seat of infection, or secondary infection by pyogenic cocci or other organism which secrete toxins having a marked general effect, that the wasting and more severe constitutional symptoms which occur in advanced cases of streptotrichosis are seen.

This general review, then, will serve to introduce the case in hand and serve as a basis for the consideration of some new points which it seems to illustrate.

C. Q., Italian, twenty-five years old. Admitted to the San Francisco Polyclinic and Postgraduate School's service at the San Francisco City and County Hospital, June 15, 1912. Died on same date. The patient was admitted with the general diagnosis of gonorrheal ophthalmia.

Past History.—Sensorium of patient entirely clear, all questions answered by aid of interpreter. Patient has been ill for about forty-five days. At that time he had an attack of right sided earache with a discharge of reddish, foul smelling pus. He gives history of repeated attacks of otorrhea since childhood, with periods of what he considered complete cessation of all symptoms.

About twenty-one days ago he had an attack of vomiting, dizziness, sweating and unsteadiness of gait. He had no chills, but he himself noted stiffness of neck. At this time he was admitted to a local hospital, where he was treated for meningitis with five injections of the original Schafer's serum without any apparent benefit. June 11th, a beginning exophthalmus was noted. The nose was examined for a suspected sinus infection, but unfortunately the ears were not examined. At this time the eye grounds were pronounced normal. The patient was discharged from the hospital the following day because of lack of funds. June 13th, patient had acute

attack of dizziness, nausea, vomiting, severe disturbance of equilibrium, followed by severe chill.

General Examination.—Small, delicately built man, unable to speak English, sensorium absolutely clear. Temperature 102,° pulse 100, full and bounding. Neck absolutely rigid. High grade of double exophthalmus. Incomplete closure of eyelids. Lies flat on back, takes no notice of surroundings, unless spoken to, and continuously moans from pain in head. Locates intense headache behind eyes and over frontal lobes.

Physical Examination.—Chest.—Loud crepitant rales on both sides.

Heart.—Sounds fairly strong, rapid and no murmurs.

Reflexes.—Very slight knee jerk right, absent left. Abdominal reflex absent. Tendon reflex absent. Kernig's sign present, positive and marked. No Babinsky.

Urine.—Slight amount of albumin, otherwise normal.

Blood.—Reds 4,500,000. Whites 45,000. Polymorphonuclear 84 per cent.

Spinal Puncture.—Fluid under great pressure, turbid, many white cells. Repeated stains show no bacteria and no growth on several media.

Examination of Eyes. (Dr. Vard Hulen.)—Marked double exophthalmus, equal on both sides. Eyes divergent. Intense chemosis, especially in lower part of ocular conjunctiva. Extremely well marked swelling between eyes, extending up over the skin of forehead. The skin is hot, red in color and brawny to the touch. There is marked pressure sensitiveness at this point, but nowhere else.

Pupils normal in size and reaction. Vision, O. D., 12/200; O. S., 8/200. Cornea clear except little haziness of both lower margins. Atropin dilates pupils evenly and widely.

Ophthalmoscopic Findings.—Extensive neuroretinitis with marked retinal edema. No signs of choked disc.

Clinical Diagnosis.—Secondary orbital cellulitis. No fluctuation of tissue around eyes. No diagnosis of intracranial complications possible from background findings.

Examination of Ears. (Dr. Henry Horn.)—Left ear normal. Right ear: Very slight tenderness over right mastoid, no swelling or edema. Some tenderness along right jugular. Right submaxillary gland somewhat swollen. External canal filled with whitish débris, small amount of pus, no odor. Ear

washed. Total destruction of drum; middle ear some smaller than usual on account of swollen mucous membrane. Upper part of external canal somewhat sunken. Weber to left. Bone conduction slightly shortened (?). Rinné negative. Hears loud shouts, with Bárány apparatus in other ear. Five minutes' irrigation with cold water produces no nystagmus and no dizziness. No spontaneous nystagmus.

Operation.—Ether narcosis, one hour and thirty minutes. Patient left table in fine shape. Pulse and respiration good. Rapid sinking and death after few hours.

The fact that the eye examination gave the impression that the exophthalmus was not due to an intracranial complication, but due to an orbital cellulitis, and the evident involvement of the sigmoid sinus and right jugular, the fair condition of the patient and the free sensorium, combined with the absolutely fatal prognosis without operative interference, were the indications for operation.

Mastoid Operation.—Usual incision. Soft parts not swollen. Few if any cells in mastoid tip involved. Sinus located far forward and only a few millimeters from the external bony canal. In spite of most careful search no antrum could be found at operation. (Its absence was afterwards confirmed at autopsy.) Middle ear filled with whitish débris, which did not give the impression of cholesteatoma, and on the other hand did not resemble any pus collection which the author had ever seen.

Sinus Operation.—Sinus laid free for an inch or more. The wall of the sigmoid sinus was entirely broken down, the clot had disintegrated and was filled with yellowish débris without odor. The thrombotic process evidently extended into the bulb and the same was rapidly and thoroughly laid free. It was seen that the jugular would have to be explored, so the uncovering of the lateral sinus was first extended about an inch toward the torcular, or until free bleeding occurred, and this was controlled with iodoform gauze tampon. The character of the thrombus changed completely as the torcular was approached, from a septic, markedly purulent mass to a reddish blood clot, which was not broken down. The free bleeding then occurred.

Ligation of the Jugular.—Incision from angle of jaw to clavicle, along the anterior border of the sternomastoid mus-

cle. Large mass of infected glands in upper third of neck had to be dissected free in order to expose jugular. Facial vein ligated, although it was not thrombosed. Ligation as close to clavicle as possible, evidently well beyond thrombus. The entire vein was thrombosed with the same reddish clot as found near torcular, and was removed en bloc. Bulb washed out from above. Upper end of vein sewed to upper angle of skin incision, after the method of Alexander. Wound closed, free drainage from lower angle.

The operative findings proved without doubt that we had to do with a far more extensive sinus involvement than was possible to correct surgically, and as the patient's general condition was good he was sent to ward. Surgical shock showed itself almost immediately, and the patient died in a few hours without recovering consciousness.

The autopsy was made through the courtesy of Prof. Wm. Ophuls, of Stanford University, the findings were dictated to his own clerk, and the subsequent microscopic work was done by him personally. I take this opportunity of expressing my great gratitude for his generosity in allowing me to use the results of his work. There was no indication at the autopsy that we had to do with anything more than an ordinary septic infection, and it was only by the thorough method which all cases are carefully worked out in the pathologic laboratories of Stanford University that the true etiologic factor in this case was discovered.

Necropsy Report.—(Prof. Wm. Ophuls). Strongly built, rather poorly nourished man, of about thirty years of age. There is a very marked exophthalmus so that the lids cannot close. The opening of the lids at present is about 1 cm. The conjunctivæ are extremely hyperemic and edematous. There are large hemorrhages around the cornea. The lids are also congested and edematous. There is marked passive congestion of the lips, of left ear, and of upper part of neck. No palpable enlargement of the superficial lymph glands. No edema. There is a slight yellow discoloration of the conjunctivæ and skin of neck and shoulders.

In back of ear there is a deep wound about 5 cm. in diameter, and extending from that posteriorly, an incision about 6 cm. long. Wound is packed with gauze. The wound extends through the bone to the dura mater, and the dura is exposed

over a large distance. The transverse sinus is opened for a distance of 1 cm., sigmoid sinus exposed to bulb of jugular vein.

On the right side of the neck there is a longitudinal incision reaching from the angle of the jaw down to the sternoclavicular joint on the right side. The wound is drained with cigarette drain. At the bottom of the wound the large vessels are exposed. The jugular vein has been resected practically for the whole distance; the upper end is sutured into the wound. One of the submaxillary lymph glands is moderately enlarged, grayish red and moist.

Skull cap normal. One of the pacchionian granulations near the longitudinal fissure extends very far into the bone, the posterior part of the longitudinal sinus has some soft clots and turbid bloody liquid. The dura mater is rather tense on both sides. Pia mater on right side shows marked diffuse hyperemia and some edema. The hyperemia is even more marked of left side. Pia mater at base is also hyperemic and edematous. The right anterior half of the pons shows a very distinct yellowish purulent infiltration; the same posteriorly, about the middle of lower surface of left hemisphere of cerebellum near the median line. On pressure upon cavernous sinus on either side there is a drop of pus discharged at its junction with the petrosus superior. The transverse sinus on both sides is also filled with thick purulent material. Same condition in the sigmoid sinus on the left side.

Soft tissues underneath the roof of the left orbit are full of pus, and there are numerous accumulations of pus underneath in the muscles. Same condition on the right side. Jugular vein on left side of neck is free. Right jugular vein is free below point of resection.

Brain normal, except for hyperemia and edema.

There is a marked atrophy of subcutaneous fatty tissue, the muscles are poorly developed. Stomach and liver in normal position. Transverse colon forms a V-shaped loop and very nearly reaches the symphysis. Few drops of fluid in peritoneal cavity.

Diaphragm.—Fifth rib both sides. Chest is rather poorly arched, long, symmetric. Heart, about three-fourths normal size. Soft postmortem clot in pulmonary artery. The heart valves are normal. Aorta is small. Many small yellow spots

in intima. The bronchi are somewhat hyperemic and contain some mucus. The pulmonary tissue is normal. There is a small superficial scar at the right apex. Right lung same as the left.

Spleen measures one and one-half times normal size. Pulp is soft, of light grayish brown color.

Left adrenal is normal.

Left kidney slightly swollen. Cortex is somewhat opaque.

Right kidney, same as left.

Pancreas, duodenum, stomach, normal.

Liver, tissue hyperemic and moist, somewhat opaque.

The rest of the aorta like the beginning.

Diagnosis.—Purulent otitis media right side (streptothrix infection). Septic thrombosis of sigmoid sinus, of right jugular, of both transverse sinuses, posterior part of longitudinal sinus, both cavernous sinuses. Phlegmon of both orbits. Double exophthalmos. Purulent meningitis. Hypoplasia of heart and aorta. Fatty spots in intima. Parenchymatous degeneration of kidneys and liver. Septic spleen. Terminal icterus. Smears were made from infected sinuses, the meninges and from pus in left orbit. They all show many partly degenerated polymorphonuclear leucocytes, few lymphocytes, many large mononuclear phagocytes. In some of the smears, especially in those from the left orbit, a moderate number of very fine, Gram positive, somewhat irregularly staining filaments with true branching. They are single or matted together in branches, mostly extracellular. No other bacteria found, in spite of intense staining and careful search.

Anatomic Diagnosis.—Purulent otitis media on right side (streptothrix infection). Septic thrombosis of right sigmoid sinus, of the right jugular, of both transverse sinuses, posterior part of longitudinal sinus, left sigmoid sinus, both superior petrosal sinuses, both cavernous sinuses. Phlegmon of both orbits. Double exophthalmus. Purulent meningitis. Hypoplasia of heart, kidneys and liver. Septic spleen. Terminal icterus.

Epikrisis.—This "autopsia in vivo et in mortuo" would seem not only to enable us to add something to the scarce literature of the intracranial involvements following this somewhat rare infection due to the streptotricheæ, but also to draw

a fairly clear picture of the method of involvement of the cavernous sinuses.

As far as a careful study of the literature goes, there has never been a case of streptothrix infection reported as having had its origin in the external ear.

The two most completely worked out cases are those of Eppinger and Löhlein. Both started as pulmonary conditions, and later by metastatic ways produced multiple brain abscesses. The infection of the great brain sinuses by this organism seems to be unknown.

The congenital absence of the antrum in our case is certainly very suggestive, and at the same time the extraordinary displacement forward of the sinus, to within a few millimeters of the external bony canal, leads one to the enticing theory that the infection of the right sigmoid sinus was by direct contiguity. This is borne out by the fact that one of the most characteristic pathologic features of a streptothrix infection is its socalled perforative tendency. If, for example, a small area of the pleura is involved, it may pass directly through the chest wall and give rise to an external abscess. With such a well known clinical tendency, we can well assume that the process started in the middle ear in early childhood, and by a slow process of ulceration broke down the few millimeters of protecting bone between it and the abnormally placed sinus, and then by the long contact with the wall of the sinus started the thrombotic process. The absence of the antrum, noted at the time of the operation and later confirmed at autopsy, would be additional proof that the process was always confined to the middle ear until later in life. The reason why the patient never received treatment for his condition was because the mastoid was never involved, and the sinus trouble was the first serious complication which he experienced.

It is also to be noted that the fever curve was at all times remarkably low for a sinus complaint of such magnitude and apparent degree of purulent degeneration, and the very few attacks of chills would seem to indicate that a streptothrix infection of the sinuses does not give rise to such marked symptoms as does a streptococcus infection. Of course, the subsequent microscopic examination with the continued absence of true bacteria clearly explains why the very turbid spinal fluid gave no bacterial content. It was of course not

examined for streptothrix at the time, as the condition was not then ever remotely suspected.

The condition of the cavernous sinus and the post- and periorbital tissues showed how impossible any satisfactory operative procedure would have been. The capsule of the orbit was so tense at the autopsy that when a puncture was made with a knife the pus spurted out under pressure. In this connection it is interesting to note how very slight were the changes observable with the ophthalmoscope.

It does not seem in place in this paper to discuss the various operative measures for attacking the infections of the cavernous sinuses. It would seem that the autopsy proves that in such a case as this operative procedure would have been worse than useless.

The autopsy findings, however, give us a certain right to theorize on the possible method by which the infection of these sinuses took place. In the diagrammatic figure shown in figure 1, the shaded parts shows the extent of the involvement. The fuller details were noted by me at the time of the autopsy for the specific purpose of tracing the mode of infection of the cavernous sinuses, and do not appear in the necropsy report of Prof. Ophuls.

The most striking thing about the appearance of the sinuses was the fact that two kinds of thrombi could be distinguished, which in all probability were different stages of the same process, but still distinct enough to show in which direction the thrombotic process was extending. First, there was the thick, yellow, semifluid purulent thrombus, which filled the right transverse, the right sigmoid, the right bulb and part of the upper part of the right jugular. About one inch from the torcular, or at the point that the operation was ended and free bleeding commenced, there was a reddish thrombus, that looked somewhat like a postmortem clot, but with greater consistency.

The purulent type extended up the right superior petrosal, did not seem to involve the inferior petrosal, invaded the sinus cavernosus and by way of the sinus circularis Ridleyi involved the whole region in a fluid purulent mass. From here the infection extended to the periorbital tissue, turning then into bags of pus under great pressure. The same purulent process now descended the left superior petrosal, involved the left sigmoid, where the purulent type was halted. The left trans-

verse and the left bulbar region were again filled with the reddish, nonpurulent clot.

It seems fair to assume then that we have to deal here with a cavernous sinus involvement of originally otitic origin, the infection extending upwards along the right superior petrosal against the stream and then downward with the stream to the opposite side.

The careful bacteriologic examination of Prof. Ophuls would seem to indicate that the infection was a pure streptothrix infection, with the addition of very few if any real pus organisms. The preoperative bacteriologic examination would seem to substantiate this condition.

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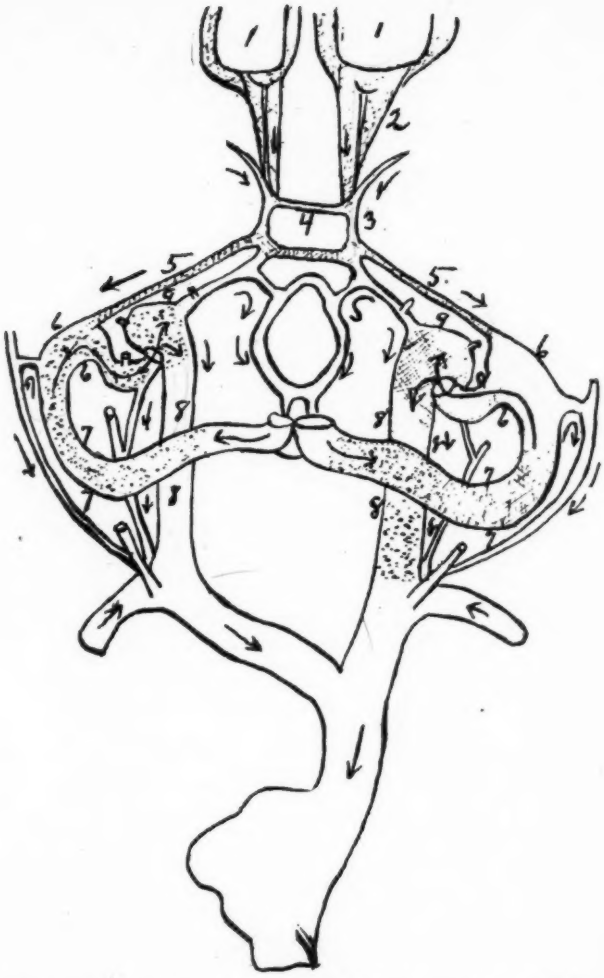

Diagram of the Sinuses. From Brühl's "Diseases of the Ear."

1. Bulbus oculi, with distended pus sac.
2. Vena ophthalmica, enormously distended.
3. Sinus cavernosus.
4. Sinus circularis Ridleyi.
5. Sinus petrosus superior.
6. Sinus sigmoideus.
7. Sinus lateralis.
8. Venæ jugularis.
9. Bulbus venæ jugularis.

LIX.

SUGGESTIVE POINTS OF ANALOGY BETWEEN OTOSCLEROSIS AND ARTHRITIS DEFORMANS.*

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London.

The literature of chronic nonsuppurative diseases of the ear and joints is very extensive, and for the purpose of considering the analogy indicated by the title of this paper, I am putting forward the summary or gist of the views held by many of the best known authorities, and place them under headings which are chosen to emphasize the numerous points or features wherein these diseases seem to have a striking similarity.

It is now the custom for many writers to regard arthritis deformans as a group of diseases, namely, rheumatoid arthritis, osteoarthritis, and spondylitis deformans. As many of the most exhaustive descriptions of chronic nonsuppurative joint affections are given under the title arthritis deformans, I have retained this form of nomenclature. It is, however, a little inconvenient, as it gives a combined picture of certain characteristics, such as the morbid changes, which some writers separate and place under the headings of rheumatoid and osteoarthritis respectively. Painter[1] calls the former atrophic and the latter hypertrophic arthritis.

The chronic nonsuppurative diseases of the ear which should be considered fully, to complete the analogy with arthritis deformans, are otosclerosis and chronic adhesive catarrhal processes, or, as Kerrison[2] calls the latter, chronic hyperplastic otitis media. As this would necessitate a much more extensive paper than the time at my disposal here permits, I have chosen otosclerosis alone to consider fully, but will refer in my conclusions to the marked analogy which, I submit,

*Read before the Sections of Laryngology and Otology, at the Eighty-first Annual Meeting of the British Medical Association, held at Brighton, July, 1913.

exists also between chronic adhesive processes and arthritis deformans.

Lucae[3] has asserted that it is not possible to separate otosclerosis and adhesive catarrhal processes clinically, but Politzer[4] differs from this view. Most present day otologists seem to be of the same opinion as Politzer, but many of the fuller descriptions of otosclerosis to be found still unavoidably mix up several of the main features of these two chronic ear affections, as is done in chronic diseases of joints.

The following are the headings under which points of analogy between otosclerosis and arthritis deformans are shown: Definition, etiology, course of disease, symmetry, trophic changes, morbid anatomy, pathogeny, and lines of treatment. Bearing upon etiology, the influence of age, sex, heredity, race, climate and other diseases and conditions, which are said to act as exciting causes of onset or exacerbations, are considered, and finally certain conclusions will be set forth.

DEFINITION.

Otosclerosis.—Politzer calls otosclerosis a progressive form of deafness, running its course without any catarrhal symptoms and presenting an entirely different character from the adhesive processes secondary to catarrh.

Kerrison gives the following definition: "The term otosclerosis is employed to describe the condition in which, independently of the health or intercurrent disease of the tympanum, the bony capsule surrounding the labyrinth is the seat of chronic nonsuppurative disease interfering with the function of hearing."

Arthritis Deformans.—Osler[5] describes it as a chronic disease of joints, characterized by changes in the synovial membranes and periarticular tissues, and in some cases by atrophic and hypertrophic changes in bones.

Keen[6] refers to it as a progressive nonsuppurative disease of joints, manifested by pain, swelling and impaired function, and leading to stiffness and deformity.

Llewellyn-Jones,[7] describing osteoarthritis, says it is a chronic oligoarticular disease of joints, the widely distributed form of which is most common in women, insidious in onset, running a slow, progressive course, the subjects of the disease being usually well nourished.

AGE.

Otosclerosis.—Gray[8] says that it is most frequent in middle life, rare under twenty, the majority of cases occurring between twenty and forty.

The commonest age incidence, according to Milligan,[9] is between fifteen and thirty.

Politzer states that it occurs especially often in elderly people, often in young healthy individuals, but those with a hereditary taint have symptoms marked at ten to fifteen.

Lake[10] gives the greatest incidence as occurring in young adult life, and states that it may begin even in childhood.

Arthritis Deformans.—The majority of Osler and McCrae's cases occurred between twenty and forty approximately. Half their cases began before the age of thirty years.

Llewellyn-Jones gives twenty to thirty as the period of greatest age incidence for rheumatoid arthritis, and says that osteoarthritis is commonest in middle and late life, but occurs in young females and even in children.

The majority of Garrod's[11] cases of arthritis deformans occurred between twenty and forty.

Painter says that rheumatoid arthritis is a disease of youth and middle life, and osteoarthritis of middle and late adult life.

SEX.

Otosclerosis.—Gray, quoting Bezold and Denker's combined cases, gives 60 per cent as occurring in women. Pierce[12] gives 60 per cent also, but Yearsley's[13] cases were as high as 83 per cent in females.

Politzer, Lake and Milligan state that females are more commonly affected than males.

Arthritis Deformans.—Painter says that osteoarthritis is more common in women than in men, but is more crippling in men, as the smaller joints are affected in women.

In Garrod's 500 cases of arthritis deformans, women were affected in 82 per cent.

In Llewellyn-Jones' 240 cases of rheumatoid arthritis, 83 per cent occurred in women. He also states that osteoarthritis is more common in females than in males.

Osler and McCrae, as well as Stewart,[14] give 50 per cent as the incidence in women.

HEREDITY.

Otosclerosis.—Bezold[15] found a history of heredity in 52, Denker[16] in 40.5, Siebenmann[17] in 35, and Pierce in 71 per cent of their cases.

Körner and Hammerschlag give a genealogic tree of five families in which forty-three cases of otosclerosis occur.

Arthritis Deformans.—Garrod's five hundred cases gave a family history of joint troubles in 43 per cent.

Osler's one hundred and seventy cases gave the same in 33 per cent, and he states that two or three children in a family may be affected.

RACE.

Neumann states that otosclerosis is much more common in the white race than in the negro, and Osler makes a similar statement regarding racial incidence about arthritis deformans.

CLIMATE AND CLIMATIC CONDITIONS.

Dryness and warmth are said to favorably influence the course of the disease in rheumatoid arthritis and chronic adhesive processes, and dampness and cold produce an injurious effect. With the exception of the benefit derived from being out of doors, climate has no influence over otosclerosis or osteoarthritis.

COURSE OF THE DISEASE.

Otosclerosis.—Lake, Milligan, Gray and Politzer say that the onset may be slow or rapid, the course progressive, though sometimes stationary for years, but may be arrested spontaneously with a fair degree of hearing.

Arthritis Deformans.—The onset may be slow or rapid, the course progressive, but in many cases, after involving two or three joints, the disease becomes arrested and no further development occurs.

SYMMETRY.

Both are usually bilateral, but one side is more advanced than the other in the slowly progressive cases. In the swiftly progressive forms early symmetry is the rule.

TROPHIC CHANGES.

Otosclerosis.—Lake gives the following: The external meatus is often large, due to change in the subepithelial tissues; the lining skin is polished, pale and devoid of cerumen; the eustachian tubes are large, with or without rhinitis sicca, the latter being frequently associated with otosclerosis. Less labyrinthine fluids or their entire absence in cases of long duration.

Politzer also mentions atrophy of the membrane, and the pale, dry condition of the external meatus, devoid of secretion.

Arthritis Deformans.—Changes in the skin and subcutaneous tissues, loss of hair, alteration in the nails, early localized sweats, followed later by cessation of this function over the affected areas, sensory and calorific disorders, wasting of muscles, cartilage and bone.

The trophic changes just enumerated are grouped under arthritis deformans by Osler and others, but Painter and Llewellyn-Jones seem to associate them exclusively with rheumatoid arthritis.

In many long standing cases of chronic hyperplastic otitis media, one finds a glossy, thin, flaccid, transparent membrane, through which the details of the inner wall of the cavum tympani can be discerned; the eustachian tubes are wide and free, and these are sometimes accompanied by a large, smooth, dry meatus, devoid of cerumen.

Are these trophic phenomena peculiar to chronic hyperplastic otitis media or to otosclerosis or to both? This question cannot be conclusively answered at present, but I think it is probable that cases of typical otosclerosis, like those of osteoarthritis, are not accompanied by any special trophic changes.

MORBID ANATOMY.

Otosclerosis.—Lake states that the earliest known change is a mucoperiostitis of the inner wall of the cavum tympani. In the cases in which the terminal lesions were examined, any or all of the following changes were found: The annular ligament had disappeared and was replaced by spongy bone, the cartilage which normally covers the footplate of the stapes and surrounds the oval window had disappeared and was replaced by new bone. Patches of osteoporosis occurred

in the labyrinthine capsule, osteophytes were found in the oval window, some of which were intratympanic and others intralabyrinthine.

Gray says that the disease begins by the absorption of bone, and the region most commonly affected is supposed to be above and in front of the oval window, and that the changes may occur in any part of the temporal bone or the ossicles or the bones of the head.

Yearsley points out that the annular ligament is widest at its anterior superior margin and that the greatest degree of movement occurs here, and that perhaps irritation and friction may be a determining cause of the greater incidence at this situation. It is interesting to note in this connection that Painter lays special emphasis on the greater incidence of osteoarthritis in the joints most commonly used by athletes.

Gray further describes areas of bone which undergo absorption by osteoclasts, and the deposition of new bone by osteoblasts, the affected areas being sharply differentiated from the normal bone. The annular ligament and cartilage disappear and are replaced by spongy bone which shows a strong affinity for staining reagents, this bone becoming dense later.

Arthritis Deformans.—The following is a summary of the morbid changes occurring in osteoarthritis as described by Painter:

The synovial membrane shows practically no change, except about spurs which form at the junction of bone and cartilage. There is no diffuse infiltration of the capsule, as in rheumatoid arthritis. The cartilage hypertrophies at the edges, spurs form by the ossification of cartilage, whose cells are arranged in columns like those of rickets, at the junction of diaphyses and epiphyses, and these spurs become dense bone later.

The pathologic changes observed by Painter in rheumatoid arthritis may be summarized as follows: The synovial membranes are infiltrated and thickened, villi form and may ossify. The involvement of cartilage is next in order of frequency, but first in the order of clinical significance. It undergoes thinning, loss of sheen, striation and erosion. The bone is last affected, there is loss of calcium content, the intratrabecular tissue is replaced by fat and fibrous tissue, the trabeculæ stand out on section and are smaller and fewer in number. There is no evidence of necrosis.

Osler's description of the morbid anatomy of arthritis deformans is briefly as follows: The synovial membranes are thickened and fringes form, the periarticular tissues are infiltrated and swollen, the cartilage becomes soft and absorbed, or may disappear and be replaced by fibrous tissue of bone. The bone ends become smooth and eburnated in long standing cases, and the shafts atrophy.

New bone may form in the ligaments, especially in the spine, and may be followed by bony ankylosis, the latter occurring commonly in the vertebral joints, but rarely in those of the extremities. Muscles undergo rapid atrophy, and a radiograph shows atrophy and hypertrophy of bone and erosion of cartilage.

Ironside Bruce[18] points out that in early cases of osteoarthritis, areas of translucency, sharply marked off from normal bone, can be seen by the X-rays.

It will be observed that Painter gives the pathologic changes of rheumatoid and osteoarthritis separately, whilst Osler gives them combined under the title arthritis deformans.

In rheumatoid arthritis the soft tissues undergo early proliferation, to be followed by atrophy, which also involves cartilage and bone. Hoffa states that bony proliferations, when they do occur in rheumatoid arthritis, are in proportion to the amount of atrophy that has taken place, and he would regard them as reparative. (Llewellyn-Jones.) In osteoarthritis the soft tissues are practically unaffected, but cartilage proliferates and goes on to ossification, with the formation of osteophytes. It has been observed that the new bone formation in cases of otosclerosis is more than sufficient to replace the old bone and cartilage which has disappeared, and no explanation is offered to account for this excess of new growth.

In my opinion the excess of new bone formation, as represented by osteophytes in any joint, is due to the proliferation and hypertrophy of cartilage at certain points, this hypertrophied cartilage becoming ultimately ossified.

The microscopic pathologic changes given by Gray in his lucid description of the morbid anatomy of otosclerosis are practically identical with the physiologic phenomena of ossification in any cartilage developing bone, from the osteoclastic and osteoblastic activity down to the greater affinity of the new bone for staining reagents.

DISEASES AND CONDITIONS SAID TO ACT AS EXCITING CAUSES OF ONSET AND EXACERBATIONS.

The following list of diseases and other conditions have been mentioned as causes of onset and exacerbations in both affections: Syphilis, tuberculosis, gout, rheumatism, rickets, anemia, tonsillitis, exposure to cold and chill, inflamed processes in the nose, nasopharynx and middle ear, puerperium, pregnancy and lactation, anxiety, sorrow, depression and nursing the sick, excess of alcohol and tobacco, trauma and arteriosclerosis.

PATHOGENY.

The exact pathogenesis of otosclerosis and arthritis deformans is unknown, but many causse have been suggested, and these may be conveniently arranged under infective, nervous, vascular, metabolic and traumatic theories of origin, any or all of which may act by producing nutritional disturbances.

OTOSCLEROSIS.

Infective Theory (microbic).—Inflammatory middle ear processes (Katz,[19] Habermann,[20] Scheibe,[21] Ballenger[22]).

Primary osteitis of the labyrinth capsule which is independent of middle ear disease (Politzer).

Syphilis, supported by Habermann, but disputed by Korner. Congenital syphilis is considered by Lake to influence onset.

Toxic.—Stucky, quoted by Kerrison, suggests that toxins resulting from errors of diet and circulating in the blood are important factors in the progress of the disease, if not in its causation.

Apparently against the infective view is the observation put forward by Gray, Politzer and Moos,[23] that otosclerosis occurs without any trace of middle ear inflammation, and that of Gradenigo as to its negative bacteriology.

Nervous Theory.—Anxiety, sorrow and depression have been suggested by Walb.[24] Walb also states that there is a greater tendency for otosclerosis to develop in individuals who are subject to mental affections and nervousness.

Vascular Theory.—Arteriosclerosis as a cause in some cases is suggested by Maupetit.[25] Habermann found the foci of the disease in the temporal bone, grouped around the blood vessels entering from the periosteum of the tympanum. Ane-

mia as a factor has been mentioned by Dundas Grant[26] and Gray, and the latter also describes areas of necrosis.

Metabolic Theory.—Puerperium, pregnancy and lactation. Bezold found otosclerosis associated in its onset with the puerperium in 18 per cent of one hundred and ninety cases. Politzer, Habermann and others have made similar observations.

Rickets (Politzer), gout (Tonybee, Buck and Argnano[27]).

Ossification of cartilage cells around labyrinth capsule (Siebenmann).

Part of a general sclerosis of the submucous tissue of the whole respiratory tract (Lake).

Traumatic Theory.—Sudden explosions, or injuries to the cranium, may increase or cause sudden deafness in these cases (Politzer).

ARTHRITIS DEFORMANS.

Infective Theory (microbic).—Organisms found in joints (Bannatyne,[28] Poynton and Payne.[29] Occurrence of influenza and gonorrhea as antecedent affections. Onset soon after tonsillitis (Bloodgood,[30] Llewellyn-Jones, Bannatyne).

Apparently against this view are the observations of Painter and Odery Symes,[31] who were unable to find organisms in the majority of the affected joints which were examined by them.

Toxic.—A toxic trophoneurosis (Tessier[32]), an autotoxemia (Tubby[33]), a cerebrospinal toxemia (Llewellyn-Jones).

Oral and alimentary sepsis have been suggested as the commonest source of the various types of toxemias.

Nervous Theory.—Worry, fatigue and depressing mental conditions. Changes found in the anterior cornua of the spinal cord (Folli,[34] Mott,[35] Bannatyne, Latham[36]). Lesions found in the peripheral nerves of joints (Pitres and Carriere[37]). The nervous theory is said by Stengel[38] and others to explain the articular changes, the muscular atrophy and the trophic disturbances in the skin.

Vascular Theory.—Arteriosclerosis is mentioned as a cause of osteoarthritis by Billroth,[39] Tessier and Lancereaux. Anemia is said to be present at the onset or during the course of the disease in osteoarthritis. Goldthwait is of opinion that the blood in osteoarthritis shows no special changes, and this view seems to be supported by the observations of Erving,[41] who made a careful blood examination in twenty cases affected by the disease.

I am not aware that this point has been fully tested in cases of otosclerosis whose appearance indicate the condition of anemia.

Metabolic Theory.—Pregnancy, lactation, puerperium and the menopause, thyroid, ovarian and uterine disturbances. Loss of calcium content in the bones, and the greater output than intake of calcium salts during the active period of the disease in rheumatoid arthritis (Painter). That the disease is due to a condition of acidosis (Barr,[42] King[43]).

Traumatic Theory.—Godlee[44] states that a severe injury in youth may determine an attack of osteoarthritis later in life. Painter points out the frequency of osteoarthritis in the most commonly used joints of athletes.

LINES OF TREATMENT.

The same or equivalent lines of treatment have been suggested in both affections.

Otosclerosis.—

Drugs.—Pot. iod., iron, arsenic, thyroid and thymus extracts, phosphorus, mercury.

Injections.—Fibrolysin, vaccines, intratympanic oils, etc.

Mechanical and Surgical.—Pneumomassage, probe massage, stapedectomy.

Counter Irritation.—Blistering, etc.

Arthritis Deformans.—

Drugs.—Pot. iod., iron, arsenic, thyroid and thymus extracts, etc.

Injections.—Fibrolysin, vaccines, intraarticular injections of sterilized vaselin (Buedinger[45]).

Mechanical and Surgical.—Massage, arthrectomy, cheilotomy (Handley and Ball[46]).

Counter Irritation.—Cautery, blistering.

CONCLUSIONS.

The following are the conclusions at which I have arrived:

1. That chronic adhesive processes of the ear, or, as Kerrison calls it, chronic hyperplastic otitis media, is rheumatoid arthritis affecting the organ of hearing.

2. That otosclerosis is osteoarthritis of the stapediovestibular articulation and of the bony labyrinthine capsule.

Dundas Grant has noted the frequent association of the two

diseases in the same individual, and the occurrence of osteo-arthritis in the female progenitor of many of the subjects of otosclerosis.

3. That the morbid changes of chronic hyperplastic otitis media and rheumatoid arthritis are practically identical, and those found in typical otosclerosis are similar to those found in osteoarthritis and spondylitis deformans.

4. That as all the affected structures are developmentally, anatomically and physiologically alike, and when in these structures identical types of destructive and reparative changes occur, the natural inference to be drawn is that these processes are the result of a common pathogenesis.

It may be urged that it is incorrect to try and establish an analogy between other articulations and that of the stapes in the oval window, but I submit that the analogy is complete from every standpoint. The periotic portion of the temporal bone is developed in cartilage around the auditory capsule, and in this respect is similar to other cartilage bones. Anatomically, the similarity is also complete. In ordinary joints of the extremities we find bone covered by hyalin cartilage, and when a joint cavity exists, synovial fluid is present. Synovial fluid, according to Halliburton, is viscid, rich lymph plus mucinoid material. It is secreted by the cells lining the synovial membrane, and its viscidity is due to the presence of mucin. In the absence of a joint cavity the bony skeletal elements, for the purpose of greater stability, are linked together by fibrous tissue or fibrocartilage, as in the case of the vertebral column in the mid-dorsal or lumbar region. (There are small synovial cavities in the cervical region between the lateral portions of the bodies.) The stapediovestibular joint is similar to a mid-dorsal articulation. The osseous labyrinth is a cartilage developed bone, and hyalin cartilage is found round the margin of the oval window and on the edge and vestibular aspect of the footplate of the stapes. The bony labyrinth walls contain interglobular cavities enclosing remnants of cartilage (Yearsley, quoting Manasse.)

The annular ligament is a fibrous structure which holds the stapes in position in the oval window. The perilymph, according to Halliburton, is a viscid fluid, not pure lymph, as it contains mucin, which is the cause of its viscidity. Physiologically, a common joint possesses the double function of being a sense organ of relative position and an organ of locomotion.

The labyrinth contains a sense organ of relative position for the head and the organ of hearing, the latter depending for the conduction of sounds on a system of joints.

5. That when, in addition to the identity of morbid anatomy, we find a striking analogy between the two diseases in the points of age, sex, race, hereditary incidence, symmetry of lesion, trophic changes, course, associated diseases and conditions which are said to influence onset and exacerbations, pathogeny and lines of treatment, the above seems to me to be the only conclusion to which an unbiased observer can come.

6. That in all the morbid changes there are two outstanding features, namely, atrophy and hypertrophy, running side by side or alternating, the atrophy being an active process as well as the hypertrophy.

7. That in the case of chronic hyperplastic otitis media and rheumatoid arthritis we find the most marked activity of the disease in the soft or clothing tissues, which first undergo thickening, to be frequently followed by atrophy, the latter ultimately involving the supporting structures.

8. That in the case of otosclerosis and osteoarthritis the most marked activity of the pathologic changes is in the firm or supporting tissues, the cartilage undergoing proliferation and ossification, with the formation of osteophytic overgrowths. The juxtaposition of hyalin cartilage and bone appears to offer the most favorable anatomic site of election at which these morbid processes take place.

If this view is correct, the included remnants of cartilage described by Manasse as being present in the osseous labyrinthine wall afford the necessary juxtaposition of the two tissues, and would explain the incidence of the patches of otosclerotic disease found scattered throughout the petrous portion of the temporal bone.

9. That the essential pathogenic factor underlying an active atrophy and hypertrophy can be only one, namely, nutritional, although the conditions which may cause nutritive disturbances are manifold.

10. That as nutritional disturbances in tissues can be brought about by infective, vascular, nervous, traumatic and metabolic influences, either or all of which may be acting in any given case, it is, therefore, impossible to fit otosclerosis and osteoarthritis into one airtight, pathogenic compartment.

I am, however, of the opinion that the essential factor

underlying the morbid changes is a chemical one, affecting the nutritive stability of developing and fully developed bone and cartilage, comparable to the rachitic phenomena which occur in developing osseous tissues, but as to how these chemical disturbances are actually initiated, it is at present not possible to explain.

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LX.

COMPLICATIONS THAT MAY ARISE DURING OR AFTER THE SUBMUCOUS OPERATION FOR THE CORRECTION OF SEPTAL DEVIATIONS.

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The operation referred to is the submucous resection after Killian, occasionally modified, but slightly, according to the indications in individual cases.

It is only after an experience of eight years, during which time about 386 cases have been operated upon, that I feel justified in writing upon the subject. I might add, too, the experience obtained in controlling student physicians in their attempts at the operation.

To me the most dreaded complication is infection, the prevention of which prompts me to outline briefly the technic employed in preparing the tables, instruments and patient, together with a description of the operation.

All instruments but the sharp cutting ones are collected and put into the sterilizer, together with a small enamel cup which is to hold the Schleich's solution. The scalpel, the swivel knife and the needle of the infiltrating syringe are put into a separate tray containing lysol solution or alcohol. Two glass-topped tables are cleansed, first with bichlorid solution (1 to 500) and then with 95 per cent alcohol. Towels are not used. The table on the operator's right hand side is to hold the instruments, the table on the left is for cotton, gauze strips, Schleich's solution, and the instruments for their administration, together with one nasal forcep to be used by the assistant in mopping or supplying mops to be used by the operator.

The patient is directed to wash his face thoroughly with soap and water, with particular attention paid to the region

of the mouth and nose, after which the face is gently mopped with alcohol. The patient's collar is loosened, a long sterilized rubber apron is put on, and the head covered with a sterile cap. He is then directed to sit on the chair between the two tables. The operator and assistant put on short sleeved sterile coats or gowns. They then scrub up thoroughly for five minutes with pumice soap and warm water, followed by a dip in alcohol. The head mirror is mopped off with alcohol, using caution to prevent the solution from getting through to the under surface of the mirror, after having once ruined a mirror by immersion in alcohol.

The assistant cuts a one inch sterilized gauze bandage into strips about eight or ten inches long, to be used as mops, and later for dressings. The nurse mixes the Schleich's solution, a powder containing morphin muriate .005, cocain. mur. 0.1, sodii chlorid 0.2, which is dissolved in 100 cc. of hot sterile water to which is added ten drops of adrenalin (1 to 1000). After two or three preliminary applications of 20 per cent cocain are made, at intervals of three minutes, the injection of Schleich's solution is begun, using a Reiner or Record syringe (capacity thirty minims), fitted with a fairly large bored reinforced needle. The first syringeful is injected submucously, as far back on one side as possible, followed by a second, 1.5 cm. above, below or in front of the first. At the beginning the solution goes into the tissues very slowly, but gradually it goes easier. Both sides are injected until all of the mucous membrane is infiltrated and the mucoperichondrium and muco-periosteum are fairly loosened from the underlying cartilage or bone. By injecting the solution thoroughly, I have frequently been able to loosen the periosteum from even sharp ridges. In the case of ridges it is well to inject thoroughly both above and below them. The fact is the injection, if well done, partly accomplishes the first step of the operation. Injections are made all the way anteriorly to the tip of the nose, occasionally swelling the soft tissues of the tip, making the nose appear pudgy. I am convinced that proper infiltration makes the operation less painful and considerably easier to perform than the mere application of a local anesthetic to the mucous membrane. It also is a factor in reducing the length of time necessary for the performance of the operation.

The nurse takes her position behind the patient, to steady

and support the head and to move it into various positions as directed by the operator. A blunt aneurism needle, which I have been accustomed to use as a hook, is introduced into the right or left nostril, depending upon the side from which the operation is to be performed. The nurse holds the hook by the upper extremity of the handle, in such a manner as to prevent her hand from coming in contact with the surgeon's or interfering in any way with his movements.

When operating from the left side the index finger of the surgeon's left hand is inserted into the right nostril, pushing the anterior portion of the cartilage to the left, while the thumb of the same hand pulls or twists the softer tissues to the right. This exposes the anterior edge of the quadrangular cartilage to the best possible view on the left side; while, at the same time, the mucous membrane is drawn forward 4 or 5 mm. from where it normally belongs; thus, an incision made through the mucous membrane at or near the edge of the cartilage falls naturally 4 or 5 mm. behind the anterior edge of the cartilage. Here I follow the plan of attempting to leave a narrow strip (2 or 3 mm.) of the anterior edge of the cartilage intact.

For the incision I use a short, wide-bellied Murphy knife, and endeavor to go through the mucoperichondrium with the first attempt, but more often it requires three or four. I do not find it necessary to curve the incision, as many operators do. Where the incision, in spite of my efforts, is too short, I lengthen it, after partial separation with the raspatory. A speculum is placed into the left side and the nostril dilated vertically, using the blades of the speculum to protect the nose against undue injury with the knife. With the speculum in place, and the assistant holding the raspatory in the pocket between the mucous membrane and the cartilage, at the same time using traction downward and lateralward, bringing the upper end of the wound considerably downward, I am enabled, with the cutting edge of the knife directed upward and medianward, to lengthen the incision 2 or 3 mm. Then changing the direction of traction, the incision is lengthened downward. The blades of the speculum stop the knife from cutting the skin on the inner surface of the nostril, an accident that is liable to occur without the use of the speculum in this way.

With the Killian raspatory (double end) the mucous membrane is separated from the cartilage and bone to or beyond

all the deviated parts. In fact, if the infiltration has been thorough, it is possible to go over the apex of sharp pointed spines and spurs without perforating the mucous membrane; of course, not always. After thorough preparation of one side the other is begun. To reach the opposite side the same Murphy knife is used to make the incision through the cartilage down to the mucous membrane of the right side, but not through it. The index finger and thumb of the left hand are applied after the same manner as was done in beginning the first incision. This dislocates the edge of the cartilage posterior to the incision to the left, bringing it into the full view of the operator, who, with the raspatory, begins to separate the cartilage from the perichondrium of the right side, following up the separation as thoroughly as on the left side. When the mucous membrane of both sides is fairly well separated the ordinary Hajek speculum is introduced, one blade on the left side between the mucoperichondrium and the cartilage, and the other blade on the right side in a similar manner. If any further separation of the mucous membrane is desired and possible, it may be attempted while the speculum is thus held. The next step is the use of the swivel knife. If the knife is at all dull, or if the cartilage tends to buckle under pressure of the knife, the incision is started with sharp pointed scissors, and followed up with the swivel knife. The instrument is pushed in an upward and posterior direction as far as it will readily go; occasionally, when the septum is deviated considerably in its anterior portion, it is necessary to tilt the knife laterally in a direction corresponding to the inclination of the cartilage. The knife is then carried in a curved direction (convexity posteriorly) downward as far as it will go without risk of engaging and tearing the mucous membrane. It is then brought forward; when, as ordinarily occurs, a fair sized (4 or 5 sq. cm.) piece of cartilage is cut loose. This is removed with a rat tooth forceps or any other suitable instrument while the speculum still remains in position. The effect of removing a large piece of cartilage at the first attempt is somewhat dramatic to one observing the performance, although the removal of a smaller piece at one time is attended, perhaps, with less risk of tearing the mucous membrane. I frequently go back a second and occasionally a third time with the swivel knife for a strip of cartilage before using the

biting forceps. From this stage on I rarely find it necessary to remove the speculum. If necessary, I mop with a gauze strip, one end of which is fed into the opening between the blades of the speculum, pushing it in firmly with the raspatory.

Further preparation of the mucous membrane of both sides is made as the necessity of the case may require. Removal of all deviated bone with bone cutting forceps (Middleton) is attempted; in some cases even to the posterior edge of the septum, and in others to the floor of the nose. I find that by keeping at it I can succeed in dissecting the inferior edge of the cartilage from the groove of the vomer with which it articulates. Finally, the mucous membrane is prepared from the upper thickened portions of the vomer on both sides. Then with a dovetailed chisel a goodly portion of it is chiseled loose, when its removal is completed with an alligator or other form of forceps.

I do not feel satisfied with my work until I am able to see the posterior pharyngeal wall through one side as freely as through the other. When the deflection requires it, I am liberal in removing the upper anterior portion of the septum; otherwise not.

At this stage of the operation the speculum is removed and the patient is directed to blow the nose somewhat forcibly; first one side, then the other, to clear out any excess of blood that may prevent me from obtaining a good view of the entire field. Besides, the effect of forcibly blowing the nose is to replace any accidentally torn or folded pieces of mucous membrane. I find that slight tearing of the mucous membrane occasionally happens with the most expert operators, and its occurrence should cause no feeling of embarrassment. I have repeatedly observed that its occurrence does not delay the recovery or cause any inconvenience to the patient, provided it is recognized and the flap properly replaced before applying the dressings. In fact, a small tear will in a measure assist drainage. In some cases I have gone even so far as to make deliberate punctures of the mucous membrane at the completion of the operation, where they have not occurred accidentally, for the purpose of affording better drainage, and with good results. The cases selected for the purpose have usually been those which I have been compelled to operate on high up, where, because of the difficulty of packing, the risk

of hematoma is increased. I do not mean to infer that I am accustomed to making accidental tears; on the contrary, it is a rare occurrence.

Before dressing the case a careful inspection of the pocket is made, after first thoroughly mopping it out with gauze, to ascertain if any loose fragments of bone remain. After satisfying myself on this point and making sure that every torn piece of mucous membrane has been properly replaced, the dressings are applied. Strips of one inch sterile gauze are carefully packed into each nasal fossa, sufficiently snug to hold the raw surfaces in close contact.

The incision wound is not sewed together. An outer dressing of gauze is applied, and over all adhesive strips to hold them in place.

I do not claim that the method here outlined is original, nor do I claim it to be superior to any of the other methods of performing the submucous operation, but the observations to be described concerning the complications and the methods employed to prevent and treat them are original.

Cocain or adrenalin poisoning may occur during the preliminary cocainization of the mucous membrane, or during the infiltration. Cocain may produce various and contradictory symptoms. I have noticed that they occur most frequently among those who were previously nervous and anxious. The symptom most commonly observed is an increase of a preexisting nervousness. The patient develops a paleness of the face and trembling of the extremities, and, when most pronounced, the patient is liable to faint; at the same time the pupils dilate slightly. It is often difficult, at least for the writer, to separate the cocain symptoms in nervous patients from the psychic disturbances that might have occurred had plain sterile water been used. I find by putting the patient on a couch in a recumbent position and reassuring him, that the symptoms pass over promptly. Alcoholics, particularly, develop loquacity and boastfulness, combined with fear. I have never observed any poisoning effect from cocain that I could truly classify as alarming. Occasionally vomiting occurs. In these cases the pulse is somewhat quickened and tension diminished. Summary of symptoms includes pallor of face and lips, tremors, fainting, dilatation of the pupils, rapid and feeble pulse, vomiting, loquacity, anxiety, followed later (five to ten

hours) by headache. I may add that amyl nitrite by inhalation is a rather effectual antidote to cocain poisoning.

There is another group of symptoms different from those already mentioned occurring, but rarely, from the adrenalin that has been added to the Schleich's solution. The symptoms observed have been rather opposite to those produced by cocain. The patient complains of throbbing sensations in the whole body, but especially of the heart. He shows symptoms of anxiety; the face becomes somewhat cyanotic and the pulse fairly rapid and of very high tension. These symptoms are of short duration (from one to two minutes), but recur with each injection of the solution. By changing to a fresh Schleich's solution minus the adrenalin, the symptoms do not reappear.

I have observed that cases which could not tolerate the least amount of adrenalin do very well on Schleich's solution alone. I might add that a careful chest examination in those susceptible to adrenalin has frequently revealed some lesion of the heart.

There is still another complication that might occur during the infiltration of Schleich's solution, of which I wish to speak, and that is air embolism. My first experience was the most startling. I had been using a syringe that was not in perfect working order. The piston was unduly loose in the barrel. As a result, it would occasionally suck in air, in spite of my efforts to avoid it. On this particular occasion about a cubic centimeter was injected, when, some seconds later, the patient developed diplopia with marked vertigo, vomiting, and some other less important nervous phenomena. The patient was put on a couch and a casual observation revealed a white tree-like formation on one side of his face, corresponding to the outlines of the facial artery and its several branches. The patient's condition improved after a half hour; however, some effects remained for two or three days. Evidently the air had gotten into the circulation through the puncture of a vein. A few years later I had a second similar experience, but fortunately the effects were less severe. I felt reasonably certain that the second case did not get any air, but of this fact I cannot be sure, for the effects corresponded, so far as the white tree formation, to that of the first case. Following close on the second experience a third occurred. This time an assist-

ant was injecting the solution into another physician, preparatory to operation. This case showed the same tree formation plus a ptosis and slight paresis of the superior rectus eye muscle, all on the right side. The effects in this case passed off entirely in about one hour. Just how many other cases may have had air emboli in other parts of the body, without apparent symptoms, cannot be determined. My object in citing these unpleasant experiences is to point out the possibility of their occurrence to those who have not yet experienced them, and to caution the beginner against the risk he takes in not seeing to it that the syringe, after loading, is free of air before injection. Furthermore, the risk of air embolism is greater when using a syringe that is not in perfect working order than one that is.

The next possible complication—or, perhaps better put, embarrassment—occurs from an incomplete or improper primary incision of the mucous membrane. It is essential that the first incision be made down to the cartilage and sufficiently long, for if not, the subsequent attempt at separation of the perichondrium will be futile.

I have watched beginners work for a half hour or more trying to separate the perichondrium, and lacerate the mucous membrane considerably because of their failure to cut clean through the perichondrium with the primary incision. I am speaking also from personal experience when I, too, was a beginner, so that I feel justified in emphasizing the importance of the first incision. If the incision has been made sufficiently deep and sufficiently long, it will not be necessary to lengthen it backward along the floor. An incision along the floor increases the risk of tearing the mucous membrane in this region. A horizontal running incision is prone to start a tear that is difficult to control. In a general way, a perforation or a tear accidentally made is of no great importance so long as the surgeon is aware of it. The only danger lies in not recognizing it immediately, for when not recognized the surgeon invariably keeps enlarging it and adding to his trouble. A very good practice is to have the patient blow the nose frequently, as this tends to replace flaps into their normal position.

After separating the mucoperichondrium in good shape for a centimeter or so, it occasionally happens that the surgeon is balked. He seems to be unable to progress farther. If he

attempts to force things, he perforates, as a rule, the same, but occasionally the other side. In those cases where the raspatory perforates the same side, the apparent obstruction has been a socalled adhesion; a condition where the perichondrium is more or less bound down to the cartilage, due, perhaps, to some old inflammatory process. Besides being more adherent, the membrane at this point is thinner and less resistant and tears readily. This difficulty might have been anticipated and guarded against by a more thorough infiltration at this particular location. During the infiltration of the septal mucous membrane I am in the habit of looking out for locations where the infiltration appears to be more difficult, and give to them more particular attention. I do not cease my efforts until the mucous membrane is found to be quite free of the cartilage or bone throughout. When infiltrating in a location obscure from view—for instance, under the ledge of a pronounced spur—the ease or difficulty of infiltration is decided by the sense of touch conveyed through the point of the needle, or by the resistance offered to the infiltration, as told by the pressure necessary to empty the syringe, or by the amount of solution that leaks from the nose when the patient puts his head forward, as he is instructed to do after each injection. If only half the quantity is lost by dripping from the nose, at least some has been retained.

In the case where the raspatory perforates the opposite side, the cause is due either to lack of experience or more often to a preexisting fracture with overlapping edges. For instance, if the posterior edge of the short anterior portion is inclined to the right, and the overlapping anterior edge of the larger posterior part to the left, it is easy to comprehend how, when operating from the left side, the raspatory, after encountering some temporary resistance, will naturally follow the plane of the anterior portion and come out on the right side of the nose. It would not be fair to term such a performance an act of clumsiness. The best means to guard against this complication is to avoid undue forward pressure with the raspatory the moment the obstacle is met. Stop and proceed to the next step of the operation in the same manner as though this obstacle had not been present. With the knife incise the cartilage, following the same direction and length of incision as the primary one—through the cartilage, but not through the muco-

perichondrium of the right side. If working from the left side, use the thumb and index finger of the left hand to dislocate the cut edges of the cartilage, and proceed after the manner already described. While separating the mucoperichondrium of the right side, when the site corresponding to the obstruction is reached, it will be found, by hugging the cartilage closely, that the instrument will jump over the posterior edge of the anterior fractured part without much difficulty. Separate the perichondrium freely in upward and downward directions, and be contented for the time being. Withdraw the instruments and proceed with the next step, the same as you would have done had no complication occurred. With an ordinary speculum, introduce one blade between the cartilage and perichondrium on one side, and the other blade between the cartilage and perichondrium on the opposite side, so that when the blades are separated the mucous membrane is protected by the blades of the speculum on each side and the cartilage is free in the middle. Push the speculum forward (into the nose) as far as it will go, without undue pressure or risk of tearing the mucous membrane. Remove, with swivel knife, scissors or punch or what you will, all of the cartilage that can be removed without injuring the mucous membrane. By so doing one will be able to remove quite easily all of the cartilage anterior to the fracture, including the overlapping portion that inclined to the right. At the completion of this step one is able to see quite clearly the right side of the remaining cartilage posteriorly, more or less free of mucous membrane, and it will be noted, too, that the raspatory follows the right surface with ease. In our earlier experience we are not able to tell immediately just where we are. We look for a free edge of the cartilage, and are surprised at not finding it. With closer inspection and by feeling with the raspatory, the inferior edge of the cartilage may be found hugging the left side of the mucoperichondrium and blending with it. This condition has been brought about by the productive (inflammatory) connective tissue, the result of the healing process that followed the original fracture, perhaps years before. After more or less manipulation with the raspatory, one is able to get between the inferior edge of the fracture and the mucoperichondrium of the left side at one point or another, when further dissection becomes quite easy.

Perhaps the most frequent complication which occurs more or less often in the experience of most of us is perforation of the mucous membrane. Unintentional perforations may occur from lack of experience (a stage through which we must all pass before we become expert); or to undue haste or recklessness, for which there is no excuse; or to certain mechanical difficulties which make a perforation nigh impossible to avert. For lack of experience the only advice is practice; more preferably on the cadaver, but where the cadaver cannot be secured, on living subjects under the guidance of an expert. For recklessness there is no advice. For mechanical difficulties the best scheme is to anticipate them and by proper infiltration try to reduce them to a minimum. The less our experience the more we are inclined to attribute our difficulties to unavoidable causes. Experience, together with proper infiltration, tends, therefore, to reduce the complications, both in number and magnitude.

One or two buttonholes in the mucous membrane need cause no anxiety, provided two of them do not occur at corresponding points of the two sides, when they are liable to be followed by a through and through perforation. A recognized buttonhole is not nearly so important a complication as one unrecognized; for, when not recognized, the operator is liable to enlarge it unintentionally. I have watched students and assistants work away trying to get past a buttonhole for a long time without success. In fact, I have been told by certain honest friends in the profession that they have worked under such conditions for a long time without success, and in disgust have given up further efforts. For this reason I feel warranted in pointing out a simple way to pull oneself out of such an embarrassment.

If for any reason a perforation has occurred during the separation of the mucous membrane from the cartilage, and the operator is sufficiently experienced to recognize it, he will find it a wise procedure not to attempt further separation at this point. Try further separation at a point above or below the perforation and get well beyond it, and then work toward the perforation from above and posteriorly, or from below and posteriorly. By so doing one avoids the risk of enlarging the perforation and at the same time avoids loss of time. Do not attempt to remove cartilage or bone with septal forceps until

a sufficiently large area about the part to be removed is separated from the mucous membrane, the object being to avoid the possibility of grasping any part of the mucous membrane with the forceps. If the operator, in spite of his efforts to avoid it, should grasp the mucous membrane at any point, he will know it when he attempts to bite or fracture off a piece of cartilage or bone and remove it. A piece of cartilage or bone free of the mucous membrane should come out easily without any resistance. If there is resistance, he may look for one of two things: either he has too large a piece of bone with a pointed edge engaged in the mucous membrane, when force is likely to cut or tear the mucous membrane during its removal; or else he has a piece of the mucous membrane within the grasp of his forceps. In the first instance a rotation in one or the other direction causes the sharp edge of bone to free itself from the mucous membrane, when the piece comes out easily. In the second instance, cease pulling, or else a good sized piece of mucous membrane will be pulled out with the bone. Remove the forceps and try to separate the mucous membrane more thoroughly, after which you may try again. If necessary, repeat this performance several times, until you are able to bite off a fragment without engaging the mucous membrane in the forceps.

Occasionally it occurs that all but the apex of a sharp spur is free, and this holds tightly to the mucous membrane, preventing easy removal.

Other physical causes that lead to tearing of the mucous membrane, after it has been freely prepared from the cartilage and bone, is the use of dovetailed chisels; especially, when chiseling in an oblique direction, as we are compelled to do in some instances. This was not an infrequent cause with me in my earlier experience. To avoid it I now shave off smaller pieces and am satisfied to go back several times, instead of attempting to chisel off too much at one time. I may mention, too, that in using the swivel knife one is liable (in certain cases where it is necessary to push the knife in an oblique direction) to engage the mucous membrane with the protruding side of the instrument and thus go through it. This error is likely to occur when one is overambitious to remove a large first piece of cartilage. It is a mistake for an operator to attempt any display of this kind, for his pride is likely to have

a fall, when he is compelled to work overtime on the balance of the operation.

Another less frequent cause of perforation is due to faulty separation of the periosteum from the sides of the articular edge of the vomer, especially in cases where the bone is thickened (osseous spine). At this point the periosteum runs up from below and follows the superior edge and down the other side, as one more or less continuous layer. When separating the mucoperichondrium from the cartilage, it will be noted that the separation progresses readily until a point is reached inferiorly, corresponding to the superior edge of the vomer. Here the raspatory balks, and if sudden force is used the membrane tears. It is never so tight at this point, however, but that milder efforts applied in the proper direction and repeated often enough will result in separation with an intact mucous membrane. A little care with sufficient patience is the only skill required to accomplish the result. Another method to overcome the difficulty is to remove all of the cartilage first, leaving only the superior or grooved edge of the vomer; then with a scalpel cut through the periosteum along the superior margin of the bone, and through this incision gradually work a separation of the periosteum from the vomer.

Excessive bleeding during the operation may be a complication of sufficient importance to cause some timid operators to abandon further efforts at the operation. Excessive bleedings are usually venous. In some cases bleeding occurs so profuse that the blood literally pours out of the nose. In other instances, though less pronounced, it nevertheless is sufficient to well up in the pocket between the layers of mucous membrane, and thus hamper the operator in getting a proper view of the field of work. The bleeding is due generally to an injury of a vein coursing near the floor of the nose. For this reason I would suggest a plan that I have long since adopted, namely, to leave the work to be done along the floor until the last, so that if by any mischance a hemorrhage should occur, let it do so after every other part has been taken care of.

Since operating in a field of blood is more or less unsafe, it is well to know just what to do under the circumstance. The plan that has usually succeeded in stilling the bleeding, in my experience, has been the tight packing of the cavity with sterile gauze for a few minutes or longer. I have found it

occasionally necessary to use in addition a little adrenalin. After the bleeding has been stilled it sometimes recurs when we attempt to take up our work again. A second or even a third pause may be necessary before we can complete the operation. In some rare cases I have completed the work satisfactorily in spite of the presence of the blood, depending upon the sense of touch; however, I do not recommend this procedure. When the operation is completed I do not let the hemorrhage worry me, even though it is quite marked, for I have found that proper packing of the nose, as regularly practiced, to replace and hold the two apposing surfaces of mucous membrane together, is sufficient to control the bleeding. Though not doubting its possibility, I have never witnessed a secondary hemorrhage, therefore cannot speak of it from experience. Slight oozing occurs in all cases after the submucous operation.

Retention of a fragment of bone or cartilage.—Lack of precaution to remove all loose fragments of bone that may have fallen into the bottom of the pocket, or that may have been unintentionally tucked away between the remaining intact bone and periosteum, is liable to lead to complications later. Though I have never known a piece to be subsequently thrown off, it is possible that such a thing might occur if the retained piece is sufficiently large. In at least one case, I believe that a long lasting localized and firm thickening of the septum, quite far back, was due to a retained fragment of bone. I would emphasize, therefore, the importance of carefully mopping out the wound cavity of blood and searching for small fragments of bone. In my earlier experience I am sure that pieces of bone must occasionally have been retained, from the fact that since having exercised extra precautions, I have been surprised to find how often I was able to detect fragments of bone located in obscure places.

Here, I am reminded of an error that I have committed on more than one occasion. It can hardly be classed as a complication, yet it has prevented me from obtaining as perfect results as were anticipated. It is a fracture of the septum near the posterior extremity. It occurs when one side has been thoroughly prepared and the other not, combined with the improper use of the septal forceps; as, for instance, using the forceps to break the bone instead of biting it off. If we

should grasp a portion of the bone that had been only partially prepared and use pressure in a lateral direction, the bone will break at the point of the instrument; and a second fracture is also likely to occur posterior to this, but in the opposite direction. Since the posterior fragment is united with the periosteum on one side, it is retained, but in a distorted position. Subsequent healing of this second fracture in a faulty position may cause a narrowing on one side near the posterior nares. On the second or third visit after removal of the dressings, an angular deformity may be observed near the posterior nares. This is fairly easy to correct by resetting the fractured bone in a more favorable position. It is better practice, however, to avoid such a fracture by using proper precautions in manipulating the septal forceps.

Faulty packing of the nose with gauze may be responsible for complications after operation. For instance, if a piece of gauze hangs down into the pharynx from the posterior nares, the patient may be bothered with a sensation of a foreign body in the pharynx, causing him to hawk to get rid of it; or if low enough to touch the base of the tongue, may cause a gagging sensation and vomiting, or even fits of spasmodic coughing. After having one experience of this kind I have guarded against it since. I found the patient quite nervous and fearful of choking. The tip of the gauze was grasped with a nasal forceps introduced into the pharynx and promptly removed without difficulty, and to the delight of the patient. Since that experience I carefully examine all cases by posterior rhinoscopy, to see that none of the gauze protrudes into the pharynx. In packing the nose I have always used several short strips (eight to ten inches long) rather than one much longer, so that in the event of a piece getting too far posteriorly it can be removed without disturbing the whole dressing.

Faulty packing of the gauze into the nose may result in getting a part of the gauze into or through a perforation and between the layers of mucous membrane, with the result that prompt healing is prevented. I have seen this occur twice, but not from my own packing. To avoid this I have the patient blow his nose—first one side, then the other—rather forcibly at the completion of the operation, and again just

before packing. With a suitable speculum, a long one is safer, introduced in such a manner that one of the blades holds the flap in place, begin to pack the nose. Insert the first strip of gauze between the speculum blades (separated as wide as the space permits), along the floor of the nose from the posterior to the anterior nares. With the nasal or dressing forceps applied over the gauze, using pressure downward to hold the gauze in place, the speculum is withdrawn. Then shift the pressure of the forceps applied to the gauze in a median direction, to cover and protect the tear, and while holding it there reintroduce the speculum; but this time with the median blade directed against the piece of gauze already in place, at the same time withdrawing the forceps. Introduce a second strip of gauze after the same manner as the first, and so on until this side of the nose is fairly well packed. The other side may be packed in a similar manner. In the average case I find that two or three pieces of one inch gauze, ten inches long, on each side, suffices for a dressing. Some cases require more and others less. Faulty packing may also result in the tucking of a torn flap inward on itself, so that more or less of the mucous surface of the flap lies directly against the raw surface of the mucoperichondrium of the opposite side. Its prevention is the same as that just described in packing of the nose in cases of perforations. If either of these complications—i. e., gauze between the layers of the mucous membrane of the two sides or tucking inward of a torn flap—be discovered after the removal of the dressings, it can be corrected by proper replacement of the flap and then holding it in position, for two or three days, with properly applied gauze.

I cannot speak of complications arising from other methods of dressing the nose, since I have had no experience with them. I have not yet attempted the method of no packing, but am tempted to at some future time, selecting a case with complicating suppurative sinusitis, where packing appears to me to be more or less contraindicated.

A very unusual complication that occurred in the experience of the writer was that of breaking a chisel in the patient's nose. The case was one with a bilateral ridge, causing the upper portion of the vomer to be very broad. A medium sized dovetailed chisel was used. A larger one would have

been better. A few firm strokes with a metal mallet drove the chisel into the bone for about a half inch. Since the bone did not yield, an attempt was made to withdraw the instrument and try at another place. As it did not come out readily, a twisting movement was made, with the unfortunate result that the chisel blade broke off flush with the bone at its point of entrance. I then attempted to remove thinner pieces, shaving the bone off, so to speak, until the broken chisel was reached. In spite of my precaution a second chisel was broken in the same way as the first. The result was that we had two pieces of chisel wedged fast in the bone, one a few millimeters above the other. A third attempt with another chisel was successful, both in removing the broad bone (thin layers at a time) and the recovery of the broken fragments of the other two chisels. My mistake was in attempting to take off too large a piece of very broad and hard bone, and in using a medium instead of a large sized chisel. Another error was that of attempting to twist the chisel to break off the bone when the bone was more resistant than the chisel. I cite this incident, hoping thereby that someone else might be saved a similar embarrassment. Aside from delaying the operation, no untoward after-effects resulted.

We shall next consider the complications that may occur after the submucous operation, but before doing so I would like to tell briefly just what I do, and advise the patient what to do at the completion of the operation.

After the gauze has been packed into the nose in the manner already described, strips of plain gauze are folded to make a pad of eight or ten thicknesses, one inch wide and about two and one-half inches long. This is put over the horizontal portion of the external nose and folded up on the two sides, and over all zinc oxid adhesive strips are used to hold the pad in place.

The patient is then told that there will occur an oozing of blood which will gradually change to a bloody serum, diminishing in amount until it ceases altogether in about twenty-four hours, and the total amount will probably not exceed two or three fluid ounces. The patient is told to expect a slight headache the evening of the operation, and not to worry about it should it occur. The contributory causes of the headache are the use of cocain, the slight shock of the

operation, and the pressure of the pack in the nose. They are also instructed to keep quiet and go on a cool liquid diet for at least twenty-four hours. In other words, even though the patient may feel good, it is better that he be treated as though he was actually sick, for by so doing he may be prevented from becoming sick. The patient is further told not to disturb the dressing nor let anyone else do so without instructions, to take no stimulants and to sleep the first night with the head high, and finally he is given aconite, one drop of the tincture every two hours, for two days. As a rule these patients do not sleep well the first night, but they do the second.

It is unusual for a patient to have a rise of temperature. When it does occur, it rarely exceeds 99.4°. In some cases the dressings are removed in twenty-four hours, in others forty-eight hours. In cases where there has been a pre-existing subacute or chronic sinusitis, the dressings are removed in twenty-four hours, when a careful inspection is made. A redressing is never applied unless hemorrhage should occur, and then only when it persists. Persistence of bleeding for ten or fifteen minutes after removal of the first dressing, calls for a pack to control it. This pack of sterile gauze is applied directly to the bleeding point. To avoid this secondary hemorrhage the gauze of the first dressing should be removed slowly until it is noticed that resistance is offered, and with the resistance bleeding. The gauze removed up to this point is cut off flush with the nose, and the remaining gauze allowed to remain in place until the following day, when it will be found to come out easily and without risk of bleeding.

If the case has done normally, after removal of the gauze dressing a small piece of sterile cotton is applied into the side of the nose where the incision had been made, and worn for a day or so to protect the wound from dust and dirt. The patient is usually able to return to his regular occupation three or four days after the operation. A normal appearing case is one which, after the removal of the dressings, shows no sign of reaction—i. e., the mucous membrane is neither too red nor swollen. In brief, the mucous membrane should appear about the same as before the operation. If the mucous membrane of the septum is red, swollen and succulent in appearance, it is an indication of infection, and the degree

of the infection is in proportion to the intensity of these signs. A slight amount of swelling clears up in a few days; a greater degree requires a longer time. In exceptional cases (in my experience, about one and a half per cent) the swelling may last two or three weeks. In these more intense cases of swelling of the septum, excessive reaction, there is a like swelling of the mucous membrane of the entire nasal cavity, and with it a seromucous discharge accompanied with fever (100° to 101°), stoppage of the nose and headache. In some of these cases I have suspected an abscess of the septum, and accordingly, after an application of cocain and adrenalin, have incised the swelling freely on both sides (making in all four or five incisions, a half inch long) without, to my surprise, obtaining pus. Some of the cases thus treated were apparently helped, while others showed no improvement whatever. In all but one of the cases showing this excessive reaction there followed a depression of the nose. I have come, therefore, to consider this complication (inflammatory reaction) as one likely to be followed by another, namely, depression of the bridge of the nose below the inferior margin of the nasal bones.

In my experience the factor that has contributed more than any other to the production of this intense reaction has been that of combining with the submucous resection an operation on one of the turbinates, especially the middle. The nasal cavity will tolerate packing fairly well, so long as there has been no particular bruising or laceration of the mucous membrane; but let there be gross injuries to the mucous membrane, for instance, lacerations, especially when they are not properly replaced; or let there be a fair sized piece of turbinate removed, leaving a large raw surface, then will packing the nose be likely to be followed by intense reaction. It is better, therefore, when doing the submucous resection, not to attempt at the same sitting any other operation on the nose, unless it is particularly indicated or of vital importance, in which event it would be well to avoid packing the nose. Infection follows operative wounds of the turbinates with decidedly greater frequency where the nose has been packed than where it has not. I have seen these infections of the turbinate so intense and widespread as to involve the entire nasal cavity. In fact, the infection of a wound of a turbinate may spread

to one or more of the sinuses, producing an empyema. So, too, we must not forget the possibility of an overlooked or latent empyema infecting a wound of the turbinate.

Another source of infection following the submucous operation is injury to the septal mucous membrane in a case where there already exists an empyema of one or more of the accessory sinuses. Here the gauze as commonly used for packing after the submucous operation bridges the way, so to speak, for the infection to travel from the exit of the infected sinus to the wound on the septum. Though I have been fortunate enough never to have had an infection following the septum operation, severe enough to cause intracranial complications and death, I can appreciate the possibility of its occurrence.

Following the submucous operation there occurs occasionally a circumscribed bilateral swelling of the septum. This swelling is more frequently due to a hematoma, i. e., an accumulation of blood between two layers of the mucous membrane. A hematoma may occur at any location, but more often in the upper anterior portion; due, no doubt, to the difficulty encountered here in applying the dressings. This complication is more or less preventable by seeing to it that the dressings are applied in such a manner as to bring a fair pressure over the entire operated portion of the septum. Another preventive measure is to puncture the septum at two or more places on each side, at the completion of the operation and before applying the dressing.

The treatment of a hematoma following the septum operation is a free incision of both sides, taking care, however, that the two incisions are not directly opposite one another, because of the risk of a permanent perforation. If the swelling is a real hematoma, it will be noted that a dark, almost black, liquid blood will issue from one or more of the incisions. The issue of blood from the incision is facilitated by the patient blowing the nose rather forcefully. Pressure and counterpressure upon opposite sides of the septum may be tried; but I have not found it necessary, since the squeezing process fails to express any additional blood after the nose has been freely blown.

A common complication of overestimated importance is a permanent perforation. The fear of it has deterred many a beginner from attempting the submucous operation sooner. By way of encouragement to him who is overanxious about

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A common complication of overestimated importance is a permanent perforation. The fear of it has deterred many a beginner from attempting the submucous operation sooner. By way of encouragement to him who is overanxious about

this complication, I would advise that he have less fear; for it is far better to have the operation followed by a perforation, than to allow a badly deflected septum to go without operation. I have come to be somewhat liberal after having had the experience of seeing six of my own cases result in a permanent perforation; otherwise, recovery in every one of these cases was prompt and perfect. One of the troublesome features of permanent perforations, when small, is a whistling sound produced by hard breathing and blowing of the nose. This may become corrected later, as the perforation tends somewhat to enlarge spontaneously. An irregular or angular perforation becomes round or oval shaped. Should the whistling noise persist, then the perforation may be enlarged by the surgeon. A case in which the mucous membrane has been torn at corresponding points on both sides, one, therefore, with a tendency to a permanent through and through perforation, is less liable to develop a hematoma or infection than a case in which a perforation has not occurred, since these perforations afford effectual means for drainage. This feature was recognized by Johann Fein, who took advantage of it and deliberately made a liberal incision on both sides with the object of obtaining a permanent perforation. Whether he still continues the practice, I cannot say.

Erysipelas is a complication about as prone to follow septum operations as any other operation. One such case was seen by the writer while doing postgraduate work. This case I recall occurred in a patient who, after his operation, sat and conversed with another patient infected with erysipelas in the waiting room of a clinic. I never learned what happened to this patient eventually, for he was transferred to a hospital for infectious diseases.

Some authors doubt the possibility of the transferability of erysipelas. Without debating the point at this time, I believe its possibility is sufficient to warrant us in isolating such cases. To avoid the risk of erysipelas and other infections, patients are instructed to keep to themselves in an atmosphere as free as possible from dust. It is well also to postpone operation of the septum in a case with acutely infected tonsils or adenoids. This is a point upon which too much stress cannot be laid. In a case with enlarged tonsils and with the history of frequent attacks of tonsillitis, though the tonsils at

the time may not appear acutely inflamed, a pack in the nose, since it compels mouth breathing temporarily, is liable to awaken an acute attack of tonsillitis from which focus infection may spread to the septum. The same applies, with even greater tendency, to the adenoids. There are patients with enlarged adenoids, who are subject to attacks of acute inflammation similar to that which occurs in the tonsils, while the tonsils may be quite insignificant. Such an adenoid can cause an infection of the septum as effectually as the tonsils; therefore, in all cases, we should inspect both tonsils and adenoids first, and when found to be enlarged or otherwise affected, they should be removed before attempting the submucous resection of the septum.

Acute empyema of one or more of the accessory sinuses is a complication that may follow in the wake of a septum operation, and, therefore, deserves consideration. Some of the cases suspected as being acute empyema following the septum operation may be acute recurrences of unrecognized chronic empyema. In the cases seen by the writer, however, the evidence pointed to their being primary acute attacks. I am reminded here of a case with severe frontal headaches on the right side, with a pronounced deviation high up, associated with polyps and pus in abundance. I decided to operate to make room for subsequent work in the region of the middle meatus of the right side. The patient had no complaint to make of the left side; accordingly, my whole attention was centered on the right side. A septum operation combined with free polyp extraction on the right side was followed by marked relief, but on the third day the patient developed pain in the region of the left frontal sinus. Careful inspection showed signs of acute and active empyema of that sinus, which lasted two or three weeks before it entirely cleared up. The occurrence of this complication following the submucous resection does not exceed 2 per cent or 3 per cent. The precautions I would suggest against this possible complication are to avoid operation during an attack of acute "cold in the head," remove diseased and enlarged tonsils and adenoids previously, and avoid combining other operations with the submucous. In some cases I have postponed operation upon the septum for several weeks. I do not mean to infer that I would delay operation upon the septum too long in the face

of a severe attack of sinusitis, for there are some few cases where the operation upon the septum may be the very thing to make room for drainage, and thus terminate the attack of sinusitis, as happened in this case on the right side.

The complication—or perhaps better, the undesirable after-effect of the submucous resection—that has grieved me the most of any, has been depression of the nose between the lower edge of the nasal bone and the tip of the nose, causing it to look somewhat flattened from the front view. Though I have been able to observe it more or less in at least 5 per cent of the cases operated (fortunately for my comfort), but one patient spoke to me of it afterward, and he was so incensed that he advised three of my prospective patients against the operation. In at least one other case—viz., one with a pronounced deviation combined with sinus disease and polyps, requiring several mechanical sittings—the patient as well as myself observed that the nose which was inclined to the left before the operation was inclined more afterward. This patient was so gratified at the results otherwise obtained that she did not put up much of an objection.

Besides infection after operation, the removal of the cartilage too far forward may act as a cause in producing this deformity. The experienced surgeon is more liable to this fault—i. e., the removal of too much cartilage anteriorly—than the beginner, from the fact that with experience we are usually more thorough and bold. There are some cases where the deviation is so pronounced anteriorly that the most favorable results, so far as breathing and ventilation are concerned, can be obtained only by the more thorough removal of the cartilage, with the risk of deformity. In those cases where the edge of the cartilage facing forward is quite straight, it is unnecessary to remove as much of it as in other cases where it is deviated.

In submucous septum operations it is my practice to remove all of the bone and cartilage that is deflected, but no more.

Infections and hematomas contribute in a measure to external deformities, and should be avoided, if possible, by taking precautions after the manner previously suggested. Relative to infections, it may be suggested by some that cleansing the nose with an antiseptic wash be tried in order to prevent

it. My reply to such a criticism is that an antiseptic to be strong enough to kill bacteria would be strong enough to injure the vitality of the epithelium, the secretion of which is normally antiseptic; and by using the antiseptic, this protective barrier is removed and a gateway to infection is opened that otherwise would have remained closed. I believe that severe infections occur more frequently when antiseptics, i. e., antiseptics in the true sense of the word, are used, than when not used.

Concerning the operation in cases that have been previously attempted, there is something to be said, since in a measure they come to us as more or less complicated cases.

One of the best authorities, Hajek, up to a few years ago, and possibly to this day, advised strongly against operating on a septum that had been previously bungled by someone else; and the more so when there has been a resulting perforation. He claimed that it is well-nigh impossible to operate them successfully. Notwithstanding his admonition, I have operated several such cases with perhaps a little less ease than others, but with just as satisfactory results. If one observes the precautions outlined in this paper concerning thorough infiltration with Schleich's solution, and the method of going past a perforation by a sort of circuitous route, he can surely accomplish his purpose. In one of the cases operated, where the nose seemed to have been literally battered up, I was able to get through with the work nicely, to my astonishment, and without even attempting it, the result was a complete closure of the perforation.

SUMMARY.

1. Operations should not be performed in the presence of acute rhinitis, tonsillitis or inflammation of the adenoids.

2. If the patient presents enlarged tonsils or adenoids, with the history of acute attacks of tonsillitis, and especially peritonsillar abscess, the tonsils should first be removed before attempting the submucous operation.

3. Operating on the septum in the presence of an acute or subacute emypema of any of the accessory sinuses should be avoided unless the patient's condition necessitates the operation for the purpose of securing drainage.

4. The previous failure of some one else, even though the

case presents a perforation, need not deter anyone with sufficient skill and courage from attempting the operation.

5. Some system of technic that will insure the least possible risk of infection from without should be adopted.

6. Strong antiseptic washes should not be used in the nose, before or after operating the septum.

7. Thorough infiltration of the mucous membrane gives more intense and longer lasting anesthesia than the local applications alone.

8. Thorough infiltration partly prepares the mucous membrane from the cartilage and bone, and thus shortens the length of the operation. It also thickens the mucous membrane so that it is less liable to tear.

9. Cocain poisoning seldom occurs, and when it does, it passes off promptly. Adrenalin poisoning occurs even less frequently, and passes off just as promptly.

10. The primary incision should be made with a sharp knife, not too far back, and as long as the space will permit. It need not be curved or made angular shaped.

11. Avoid, if possible, lacerating the mucous membrane, and if, in spite of precautions, a tear should occur, do not let it worry you overmuch.

12. In the event of a tear high up, plus an empyema, it would be better not to use gauze dressings, for in that region the gauze will act as a carrier to convey the pus to the wound.

13. All crooked portions of the septal cartilage and bone should be removed.

14. The removal of too much cartilage anteriorly and high up, when this portion of the septum is fairly straight, should be avoided.

15. In using the chisel near the floor of the nose, small pieces should be removed rather than to attempt too much, because of the risk of breaking off the chisel in the hard bone.

16. In the event of an unintentional fracture far back, the fractured portion should be removed, or if it is not denuded enough to do so, on account of one side being attached, the fracture should be reduced.

17. In packing the nose the gauze or whatever is used should be applied with even, fairly firm pressure over the entire mucous membrane of the two sides, corresponding to the area from which the bone and cartilage has been removed.

18. After operation the patient should be given explicit directions as to what he is to do and what not to do. In fact he should be treated in as strict a manner as one would after any other operation of like magnitude.

19. After removing the dressings, a bilateral diffuse swelling, with some fever, indicates an infection, which is to be treated accordingly.

20. A bilateral circumscribed swelling of the septum speaks for a hematoma, and should be freely incised from both sides.

21. Where a through and through perforation is found after operation, it should be left alone. If it is small and the patient finds a whistle in his nose, the perforation may be enlarged.

22. A rise of temperature with pain or headache calls for removal of dressings, for the probability is that there is an infection, and the sooner the dressings are removed the less severe the infection is likely to be.

23. Too much should not be attempted at one sitting. If the submucous operation has been performed, additional work upon the turbinates should be postponed. Removal of polyps may occasionally be attempted.

24. If some deformity occurs after the operation, in spite of every precaution, the only thing that can be done is to trust that the patient's improvement otherwise overbalances it. Apropos of the subject of slight deformities, let all conscientious surgeons be liberal enough to defend one another.

In conclusion, I wish to say, that in spite of all its drawbacks, which when put together appear so immense, I believe, sincerely, that the submucous operation of the septum is the most uniformly satisfactory operation that we as rhinologists are called upon to perform.

LXI.

APHTHOUS ULCERATION OF THE UPPER AIR PASSAGES IN PULMONARY TUBERCULOSIS—A RETROSPECT AND A REVIEW.*

By John N. Mackenzie, M. D.,

Baltimore.

There is perhaps no subject in the whole domain of laryngeal pathology which has excited such universal interest and awakened so much animated discussion as the pathologic conception of laryngotracheal phthisis. The association of ulcerative disease of the upper air passages with pulmonary consumption has been recognized from an early time, and, although over a century has elapsed since the publication of Petit's little classic,[1] the most divergent opinions have prevailed concerning the correct interpretation of their reciprocal relationship. The older writers described only one form of ulceration, and busied themselves with the question of a laryngeal phthisis, in the generic sense of the term, without making those finer histologic distinctions which pathologic anatomy later developed. The existence of true tubercle as the chief etiologic factor in the production of what is commonly known as phthisis laryngea cannot be gainsaid, in the light of pathologic investigation, and it now only remains to be decided whether all forms of ulceration of the upper air passages which are encountered in the course of pulmonary tuberculosis are to be referred directly to the tubercle bacillus as their primary cause, or whether, on the other hand, there are not some which may be regarded as owing their origin to influences associated with, but not necessarily dependent upon, the true tubercular process.

I propose in this paper to consider a variety of ulceration which has an important bearing on this question, and which,

*Read before the American Laryngological Association, Atlantic City, N. J., May 9, 1912.

on account of its more frequent localization in the lower portion of the trachea and bronchi, and its usual occurrence in the later stages of pulmonary tuberculosis, comes more within the notice of the pathologic anatomist than under the eye of the clinical observer. It is probably due mainly to this fact that it has been overlooked by many of the leading writers on diseases of the throat, notably in England and America, and its existence lost sight of in the discussion of more pressing and therefore more engrossing questions.

This, the socalled aphthous erosion of the older writers, was first depicted in the atlases of Cruveilhier[2] and Albers,[3] and clearly distinguished from tubercular ulceration by Rokitansky.[4] In his Lehrbuch der pathologischen Anatomie, Rokitansky calls attention to round or oval erosions of the mucous membrane, surrounded by a scarlet border and covered with a yellowish slough, which are found, during the course of pulmonary phthisis, in great numbers in the trachea, and which, after the extrusion of the slough, coalesce and lend to the membrane a sieve-like, worm-eaten appearance. These, which are not to be confounded with tubercular ulceration, are found, according to this observer, in greatest number on the side of the trachea corresponding to the lung whose disorganization is the more pronounced.

Föster,[5] who also clearly recognized this peculiar form of ulceration, whilst admitting its aphthous nature and pointing significantly to the fact that it may resemble tubercular ulceration so closely as to render an appeal to the microscope necessary to differentiate them, asserts as the result of his observations that the majority of the socalled aphthous erosions of the trachea are in reality tubercular. On the other hand, many authors look upon these ulcers as catarrhal (Louis,[6] Trousseau,[7] Andral,[8] Hasse,[9] Rheiner,[10] Rühle,[11], Heinze,[12] and others), or, as Rindfleisch has done, refer them to follicular ulceration; whilst their tubercular nature is maintained by Isambert,[13] Ziegler,[14] Eppinger,[15] and others. Eppinger, who describes them as tubercular arrosions, held, in the absence of absolute histologic proof of direct tubercular infiltration, that they are, notwithstanding, specific tubercular products, and suggested that they may be evoked through the operation of the monas tuberculosum of Klebs.

Many years ago I examined the respiratory organs of all

patients dead of tuberculosis in the Rudolph and Jewish hospitals of Vienna during a period extending over five months, and published the results of the examination of this abundant material in the *Monatsschrift für Ohrenheilkunde*[16] in Berlin.[17] I wish today to present to you a description of this aphthous erosion, based upon an analysis of these cases, and to attempt to define, as accurately as possible, the relations between it and pulmonary tuberculosis.

Macroscopic Appearances.—Aphthous ulceration appears as small, for the most part superficial, irregularly round or oval erosion of the mucous membrane, which vary in size from a pin's head to a small pea, and which, separate and distinct, stud the surface of the membrane, or, coalescing, form a large ulcer, which spreads in an irregular manner over the mucous surface. Commencing as superficial erosions of the mucous membrane, with tolerably defined margins, they assume later a more characteristic appearance; their walls become sharp, irregular, and somewhat elevated, and vary in color from a light reddish shade to a pronounced livid hue. The base is either smooth and red in color, or presents a rough, uneven appearance. It is generally covered with a yellow slough, or with a more or less orange colored exudation. The ulcers are surrounded by a brilliant scarlet border, and the mucous membrane between them is slightly swollen and deeply hyperemic, thus forming a pathologic picture of great beauty. The ulcers, which are always multiple and never single, are most frequently met with, and are by far more abundant in the lower portion of the trachea and bronchi; in the former the posterior wall seems to be their most frequent seat, though the whole mucous membrane may be covered with them. I have seen the trachea in its entire length and circumference, together with the bronchi of both sides, almost completely destroyed and its mucous membrane converted into a necrotic mass. In my experience they are never found in the bronchi of the unaffected lung, but where both lungs are disorganized, the bronchi of both sides are studded with them, and in most cases they can be traced directly into the cavities themselves. They are met with less frequently in the larynx and the pharynx. In the former they select the laryngeal aspect of the epiglottis, the anterior surface of the arytenoid cartilages and the interarytenoid fold as their favorite seats. In the pharynx I have seen them most

frequently in the pyriform sinuses, where they sometimes assume a considerable size. Their occurrence here is most probably explained by the accumulation in these cavities of the irritant sputum. In the larynx they lose to a great extent their characteristic appearances and approach more closely the type of simple catarrhal ulceration. They are sometimes found associated with or in the neighborhood of tubercular ulcers, and I have seen them more frequently than tubercular ulceration in the trachea.

Microscopic Appearances.—The sections presented in general the typical picture of circumscribed necrosis of the mucous membrane. At those situations where sloughs were present, the epithelium and upper layers of the mucosa were converted into a finely granular detritus, and the neighboring connective tissue of the mucosa, and also, to a great extent, the submucosa, were highly infiltrated with lymphoid cells. This round cell infiltration invaded the tissues between the mucous follicles, without, however, involving them in a marked degree in the inflammatory process. The spots where ulceration had already occurred presented in the base and edges of the ulcer likewise a granular detritus, and the surrounding tissues were also the seat of a high grade round cell infiltration. Here and there it was possible to recognize transition stages of both the above described processes; viz., nodules, so to speak, of necrobiotic tissue, in which the slough had already begun to separate, although adhering closely at several points. The examination of a great many sections showed that the first stage of the aphthous process consisted in a high grade round celled infiltration of the mucous membrane in its upper layers, with cloudy swelling of the superimposed epithelium. Distinct miliary tubercles were nowhere to be seen, neither within nor in the neighborhood of the necrobiotic nodules. Sometimes individual nodules coalesced, and especially was this true of the bronchi, so that the membrane over a considerable area presented the above described sloughing of the upper layers of the mucosa. The above anatomic appearances, therefore, leave no room for doubt that these ulcerated areas are the result of a circumscribed superficial mycotic or septic inflammation of the mucous membrane—that is to say, an infiltration of its tissues with so rich and rapid cell proliferation as to eventuate in necrosis and sloughing of the superficial layers.

In view, then, of this interpretation of their nature, what practical conclusions can be drawn concerning the etiology of these lesions? Are they related to the tubercular process in the lungs, and, if so, what is the nature of the relationship? The first question I must answer decidedly in the affirmative, since they are most frequently found in connection with well pronounced tubercular changes in the lungs; and in regard to the second, it seems impossible to resist the conclusion that the foul contents of the pulmonary caverns, lingering in the bronchi and lower trachea, so irritate the mucous membrane as to cause it to react with tissue necrosis.

It may be here remarked that in years gone by two opinions prevailed concerning the part which the sputum plays in the production of laryngeal phthisis. Following Louis,[18] the advocates of the one affirmed that the sputum and ulcers stand to each other in the relation of cause and effect, although they differed widely as to the nature of the ulcerative process, some maintaining it to be a simple catarrhal erosion, while others regarded it as tubercular, evoked through the invasion or infection, as it were, of the superficial layers of the mucosa by the micrococci in the expectorated matter (Ziegler, Eppinger). The champions of the other and perhaps more popular idea were equally confirmed in their belief that the sputum played no role whatsoever in the destructive changes, and some even went so far as to deny the possibility of corrosion of an intact mucous membrane by the contents of the pulmonary caverns (Heinze).

Passing then to a summary of the evidence upon which the corelationship between aphthous as contradistinguished from tubercular ulceration of the air passages and the corrosive action of the sputum rests, the following facts present themselves in favor of the view expressed above:

(a) The predilection of the ulcers for those places which are in constant contact with the sputum, viz., the trachea and the bronchi, and especially the posterior wall of the former and the laryngeal surface of the epiglottis.

(b) The fact that they increase in number as the lungs are approached, and that they are much less common in the upper portion of the trachea and larynx. It was these familiar facts that suggested to Louis the hypothesis which bears his name.

(c) That they may be traced from the bifurcation of the

trachea to the division of the bronchial tubes where in all cases they become visible only in one tube, which is that leading to a cavity. This remarkable distribution of the ulceration in pulmonary tuberculosis, which I have repeatedly verified by dissection, did not escape the observation of Stokes, who advanced it in favor of Louis' hypothesis.

(d) Their absence in the bronchi of lungs which are not the seat of cavities or advanced tubercular change. This, at least, has been my observation, and I am unaware that they have been seen by others when the lungs were not the seat of pronounced disorganization.

(e) Their occasional presence in the esophagus, stomach and intestines from swallowing the sputum. In one of my cases ulceration was found in the esophagus and small intestine macro- and microscopically identical with the aphthous ulcer in the larynx. Tubercle was present only in the lungs.

(f) Finally, their occasional appearance in suppurative pneumonia (Wunderlich[19]) and gangrene of the lung.

These lesions, therefore, may be regarded as the result of an inoculation, so to speak, of the mucous membrane with the detritus from the broken down pulmonary tissue, leading to the formation of a loss of substance pathologically distinct from, but possessing some of the characteristics of, the tubercular ulcer. Notwithstanding, however, that these nodules owe their existence to the disintegration in the lung, it does not seem to me justifiable to look upon them, as Eppinger has done, as specific tubercular products, for the following reasons:

First—That they differ histologically in no respect from the circumscribed mycotic or septic ulceration of the mucous membranes of individuals in whom no evidence of tuberculosis exists; an important point in their differentiation from the well defined and characteristic anatomic changes of tubercular infiltration and ulceration. In this connection, it may be added that the tubercular process, as shown by Heinze, commences under the epithelium, whereas in the ulcers under review that structure is always first affected.

Second—That well pronounced circumscribed inflammatory infiltration resulting from necrobiosis and sloughing and histologically identical with these aphthous lesions is found, although not as frequently and as abundantly, in the mucous

membrane of the air passages, and particularly in that of the trachea, as a secondary complication in diseases other than pulmonary tuberculosis. Thus, in circumscribed gangrene of the lung, or as the result of perforation into the trachea of broken down, sloughing bronchial glands, small, round "aphthous" erosions may be detected in the mucous membrane of the trachea and bronchi, which are the histologic analogues of those described above.

Third—That on other mucous membranes of the body, under similar conditions, namely, as the result of a neighboring long standing gangrenous process, or the constant passage over them of an ichorous discharge, ulceration is found, identical in every respect with that described above. This is true, for instance, of the mucous membrane of the vagina in gangrenous conditions of the uterus. In one of my cases of this kind the vagina was studded with small aphthous erosions, the result of gangrene of the portio vaginalis uteri from an unknown cause.

The great number of the ulcers, their absence in intense, acute and chronic simple catarrh of the pharyngobronchial membrane, their characteristic microscopic appearances, especially their often elevated, livid, irregular edges and sloughing base, and the picture which the microscope reveals, are sufficient to exclude the idea of their catarrhal nature, and strongly militate against the hypothesis which would relegate them to the simple inflammatory condition of the mucous membrane which often ushers in or accompanies the tubercular process in the air passages.

I think, therefore, in conclusion, that my observations go to show that aphthous ulceration of the larynx, trachea and bronchi occurring in the course of pulmonary tuberculosis is related to the affection in the lung in so far as it is produced by the corrosive action of the sputum; but that in the present state of our knowledge (1880) it is impossible to affirm its specific tubercular nature.

At the time these observations were made our study of the subject was rendered infinitely more difficult than it is at present by our comparatively scant knowledge of its bacteriology, and the technic of examination then in use was ridiculously primitive when compared with the methods of precision and exactness of modern scientific research. Moreover, the darkness surrounding the investigation of the disease had not been

illumined by the isolation of the tubercle bacillus, and we had therefore to work out the problem without the light of that great discovery.

For these reasons it is not surprising that in days gone by there should have been so much divergence of opinion in regard to the ulceration under consideration. But even with modern histologic and bacteriologic methods of investigation there was still no consensus of opinion among authorities on the subject until the atmosphere of the question was cleared by the painstaking and interesting researches of Ziba, in the Pathological Institute at Strassburg.

Before referring to this important work permit me to pass briefly in retrospect and review the opinions of some of those who have specially investigated the subject histologically during the past thirty years, and who from the carefulness of their studies are entitled to eminent respect.

While Virchow[20] admitted the possibility of "erosive" ulceration of the larynx and trachea in tuberculosis, B. Fränkel,[21] on the other hand, maintained, from the presence of tubercle bacilli in the secretion obtained from them, that the erosions in question were true tubercular products. Schech,[22] while acknowledging that he was repeatedly unable to find either tubercle or bacilli in the walls and base of the ulcer, nevertheless declared that it is a specific tubercular lesion and sought to explain the frequent absence of characteristic structure and bacillus on the ground that at the time of the examination the tubercles had already been cast off or were lost to sight in the overwhelming multitude of the lymphoid cells of the secretion. Heryng,[23] as the result of numerous bacteriologic examinations, agrees with Schottelius that simple erosions of the mucous membrane of the larynx may exist which are due to the secretion from the lung, and which may occur quite apart from infection by the tubercle bacillus, but which can subsequently be secondarily infected with tuberculosis.

Orth[24] believes that the vast majority of the socalled "aphthous," "erosive" or "corrosive" losses of substance are true tubercular ulcers. At the same time he calls attention to the fact that the sputum of the cavities contain, often in considerable number, bacteria other than the tubercle bacillus, and that furthermore, chemical changes may occur in the secretion which may exert a corrosive or septic or necrotic influence

quite aside from that produced by the specific poison of tuberculosis.

Eugen Fränkel[25] concludes from his histologic and bacteriologic researches that all destructive lesions of the larynx occurring during pulmonary tuberculosis are due to the operation of the tubercle bacillus, which invades the tissues through the epithelium, and is often assisted in its destructive action by pyogenic organisms. This observer admits, however, that there are some cases of ulceration which cannot be regarded as specific tubercular products, since they show neither the characteristic anatomic changes of the disease nor the presence of the bacillus. He would set these cases aside as examples of mycotic epithelium necrosis due to invasion of pathogenic cocci through the surface. This view of Fränkel that the overwhelming majority of larynx ulcers in consumption are true tubercular in origin is shared by most pathologists and laryngologists, with the exception perhaps of Gottstein,[26] who regards the socalled aphthous ulcer as the result of simple necrotic sloughing of the epithelium.

In talking the matter over with Professor Hans Chiari in the autumn of 1910, when he was in this country to deliver the Herter lectures at the Johns Hopkins University, he was very positive in his opinion that the work done by me over thirty years ago was good today; but that it would have to be gone over again with modern methods of examination, and in the light of the discovery of the tubercle bacillus; and he furthermore said that on his return to Strassburg he would have it done.

The following spring the work of Ziba,[27] inspired by Chiari, appeared, which, I venture to think, has shed much light upon the subject, and which is important from the fact that the author has approached the question in a different and more comprehensive and liberal way than that pursued by some other observers. For a detailed account of his observations and methods I must refer to the original publication, and content myself simply with a summary of his conclusions.

As the result of his histologic and bacteriologic researches he believes with me in the existence of the aphthous ulcer of the larynx and trachea in pulmonary tuberculosis, as contradistinguished from the true tubercular ulcer. He has found that the aphthous ulcer is caused by streptococci in the

sputum from the lung cavities—that infection takes place from the inner surface of the mucosa and produces an inflammatory process which is associated with tissue necrosis, and which leads to the formation of the aphthous ulcer—the latter may later become tubercular, but in the aphthous ulcer originally there is neither in its base nor walls any trace of miliary tubercle. This conception of its nature is, furthermore, supported by bacteriologic examination. Only in those ulcers in which miliary tubercle was discovered microscopically were tubercle bacilli found, and solely in the region of the miliary tubercle itself. In all these cases both miliary tubercle and tubercle bacilli were absent. Even in those cases in which tubercle bacilli were found in the secretion which covered the mucous membrane, both characteristic structure and bacillus were wanting.

Aphthous ulceration of the larynx and trachea produced by streptococci may invite the development of tubercular ulceration. This is all the more probable when it is remembered that it appears in greatest numbers in the last stages of pulmonary tuberculosis. At this period the great majority of the ulcers are simply streptomycotic in character, and secondary tuberculosis develops in them only here and there.

I have brought this question before you for discussion reluctantly and with much diffidence on account of my personal interest in the matter. But, quite apart from any such consideration, the subject is deserving of attention in view of the fact that it is overlooked by nearly all English-speaking authors on tuberculosis of the upper air passages.

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LXII.

IMMEDIATE AND REMOTE SEQUELÆ OF RADICAL REMOVAL OF THE FAUCIAL TONSIL—OBSERVATION OF TWO HUNDRED CASES.*

By Virginius Dabney, M. D.,

Washington.

When we reflect that operation looking to the removal or reduction in size of the tonsil was devised and practiced in the middle ages, the amount of literature on this subject in all its phases is in nowise surprising, great though it be. Hence, a few only of those articles pertinent to the limited scope of this paper will be quoted here. Similarly, the facility with which any point of view could be sustained by reference to these authors is obvious, but it is one thing to state a fact and quite another to account scientifically for its existence. Thus, the statement of sterility in the male or female who has had a tonsillectomy done before puberty, the occurrence of blindness and other calamities of physical origin, all may have existed, but such post hoc, propter hoc reasoning cannot convict the laryngologist.

In order to narrow the field of this paper the radical removal alone is considered, which can, of course, be done by as many different instruments as methods; obviously, again, the partial and the radical method may share in common some of the sequelæ, notably hemorrhage and injury to the adnexa. Consequently, I do not wish to be understood as limiting any given result to the radical method merely because I mention it in that connection.

The agitation to stay the hand of the laryngologist in his alleged ruthless onslaught on the innocent tonsil is timely, if a little overdone, insofar as it has directed attention to the harmful effects that may and do follow the removal of these problematic organs. This plea for considerate surgery is emi-

*Candidate's thesis, American Laryngological Association.

nently proper, but the lay press has made such spectacular use of it that a legitimate operation, even where it is vital to the patient's health, is now performed under great protest on the part of parents who have had their opinions moulded by the public prints.

PRIMARY AND SECONDARY HEMORRHAGE.

This sequela is given prime consideration by all observers, though a few seem to regard it as not especially serious (Freer, Richardson, Ballenger), from which view I must sharply dissent. Although I have never had nor seen a fatal hemorrhage following an operation on the tonsils, I have been profoundly alarmed at several of my own cases, and have seen some that resisted the skill and dexterity of experienced operators. The sinister and disconcerting fact is, in my mind, that patients have died from this cause. A colleague told me of a fatal case he had had in which the patient, a young girl of about fifteen years of age, swallowed quietly the steady ooze from the tonsillar cavity till she was exsanguinated. Her pallor finally attracted the attention of a nurse, who gave the alarm, but it was too late. This case had had nothing approximating a hemorrhage at operation, and there was no reason to fear any complication, though she really died of neglect on the part of the ward nurse, who should have watched her carefully.

E. A. Crockett, in stating that he considers hemorrhage a serious matter, says that in a year and a half twelve deaths from this have been reported in and about Boston, leading him to infer that there are many more than that number. However, in his discussion he brings out the fact that only one in every group of twelve untoward sequelæ of the tonsil operation was noted as occurring at the hands of a competent operator, meaning either a laryngologist or an operator who had had experience and training in this work. In addition to the twelve deaths referred to, he records "many serious hemorrhages," one requiring ligature of the common carotid after a scissors enucleation, and two deaths in two minutes, following a sharp dissection with the scalpel; truly an astounding calamity, absolutely inexcusable. None of these cases were his own, though he lost one patient from ether pneumonia followed by empyema, acute mastoiditis and meningitis.

Lindley Sewell was able to find reports of nineteen deaths,

and fifty severe hemorrhages which were controlled. It is his opinion that the menstrual period conduces to greater loss of blood, if the patient be operated upon at that time. I have not been able to observe any difference, though I have repeatedly operated at this period and made careful notes on every case, in order to determine this point.

Ballenger, in nine thousand cases, had only two hemorrhages severe enough to cause marked anemia and exhaustion. Pfingst observed a hematoma in the pharynx as a result of suturing the pillars over a pledget of gauze to control a hemorrhage which began eight hours after operation. Cassellberry thinks that about one case in every seventy-five caused him some difficulty in stopping the flow of blood, out of an experience represented by fifteen hundred cases.

The case which caused me much anxiety was the result of a punch operation, coming on three hours after he had left my office. The flow was controlled with lumps of ice, fitted snugly into the bleeding cavity. Ten days later, while walking slowly on the street, his throat began bleeding and did not stop till he had fallen from syncope. I have seen twice in consultation an apparently uncontrollable primary hemorrhage due to failure to clean out thoroughly the supratonsillar fossa. A hard, fibrous nodule containing bleeding vessels was in each case discovered, and its snaring immediately stopped the flow of blood. I have observed that there are three points whence stubborn bleeding may arise in the radical operation: the supratonsillar fossa, the middle third of the posterior pillar, and the point where the infratonsillar fossa blends with the pharyngeal wall. In the method which involves divulsion of the loosened and hanging tonsil, there is apt to be a stripping of the small vessels in their length, which will always cause a type of bleeding necessarily stubborn, for obvious mechanical reasons. I have had three cases which required suturing of the pillars to control secondary hemorrhage, and innumerable ones in which I deemed it wiser to crush bleeding points or areas with powerful forceps rather than try pressure, though the latter method would have in time controlled the trouble, I feel sure.

Stucky reports a death from exsanguination a few hours after operation, and nine cases of secondary hemorrhage which required a second anesthesia and ligation or suturing the pillars. He does not give the names of the operators, or any particulars of the methods employed at operation.

C. W. Richardson, in a very frank and comprehensive paper on complications of the tonsil operations, reports no serious bleeding in his own cases, which he attributes to his technic of finger enucleation. In this I think he is correct, though I do only a partial finger separation and finish with the snare.

HYPERPYREXIA.

Richardson and Wishart both report a fatal case with this complication, in which the temperature rose to 107° F. The former case had a temperature of 99.4° F. at operation.

To be classed under this head, perhaps, is the case of a chauffeur, of good habits, whose tonsils I removed under the severe handicap of a badly given anesthetic. He was never sufficiently relaxed to dispense with the orderly's help, and the hemorrhage, from two torn vessels on the posterior pillar, rendered my task particularly difficult throughout. From normal at operation his temperature in two hours rose to 102° F. and in four hours to 105° F., and he was in a mild delirium for twenty-four hours. His chest, urine, blood and history were all negative. By lysis, in five days his temperature became normal, but it was a month before he regained his strength, though an unusually muscular man. All temperature readings were by axilla, which would show more fever than indicated by the figures 105° F., of course.

INFARCT OF THE LUNG.

Richardson reports two cases of this rare complication, in one of which it subsequently became necessary to open and drain a lung abscess. He also describes a case reported by Le Play, where the resulting abscess was probably due to an embolism.

SEPSIS.

It is probable that every case of tonsillectomy is complicated by some local infection, which is a natural consequence of laying bare two large granulating surfaces, with some trauma, in the presence of numerous bacteria. Certainly the three cases of cellulitis, one with the further issue of facial paralysis of five weeks' duration, reported by J. A. Stucky, would suggest this contingency. I have had five cases where swelling of the

glands in the parotid region was sufficiently marked to require attention, though none went to suppuration. The presence of edema, which I have observed in at least 50 per cent of my cases, and have heard colleagues mention as a frequent complication in their work, however, is due to infection only in part, I believe, and has never caused any serious trouble in the convalescence. The removal of the tonsil in toto is likewise the removal of the support of the pillars and velum; hence, the relaxation and venous stasis contribute to the edema, if they do not actually cause it. The flabby, immobile palate allows food and liquids to pass into the posterior nares, simulating paresis; in fact, Sewell reports two cases of paresis of the soft palate which suggest to me merely a severely relaxed, edematous palate, and a patient naturally unwilling to incur great distress by voluntarily putting on the stretch such sensitive parts. Mild general sepsis is fairly common, accompanied by a temperature of 100° F., but severe cases of systemic infection, involving gangrene of cervical muscles, or septic phlebitis with optic neuritis and delirium, such as reported in Richardson's paper, as seen by L. W. Dean, are fortunately rare. Thus, Ballenger describes but two cases of his own, both due to the streptococcus. L. C. Deane says about 2 per cent of his cases showed this effect. Bauchacourt, in Dean's paper, reports one case of edema of the glottis with death, and Martin two deaths, while Dean gives one death from septicemia in his own practice. This occurred on the sixth day.

EMPHYSEMA.

Richardson's case, in which the air entered the subcutaneous area within an hour and cleared up in forty-eight hours, and the case of B. D. Parrish, where the emphysematous infiltration began a few minutes after operation, and extended from the right eyelid down the cheek, face and chest to the last rib, are the only cases I find recorded. I am indebted to Dr. Richardson's paper for these.

PNEUMONIA AND PLEURISY.

Scattered through the literature there is to be found an occasional report of pneumonia following a tonsillectomy, but as it is not always clear to the reader whether the death was due

to the ether or to inspired matter, it is not easy to classify them. However, Richardson reports one pneumonia which proved fatal, and two pleurisies, neither of which was fatal. According to Crockett, there have been in recent years but two ether pneumonias at the Massachusetts Charitable Eye and Ear Infirmary, one of which died, but he has collected reports that show in the Boston neighborhood one death, which he says was due to strangulation from vomitus, and one death he calls an anesthetic death, though suspecting an enlarged thymus. A death from such a cause can in no way be attributed to the operation of tonsillectomy per se, but it is reported in this connection by the following, each of whom had one fatality: Dench, T. J. Harris, F. R. Packard. I have had one case of pneumonia which was certainly due to the manner of administration of the ether. His convalescence was very slow, and his temperature lasted for three weeks, but he suffered no permanent detriment.

RHEUMATISM.

Three days after removing the tonsils of a young boy, a slight swelling was observed on the outer aspect of each ankle, and a temperature of 100° F. This subsequently rose to 101.2° F., and the case ran a typical course and history of acute articular rheumatism, with involvement of the knees as well as ankles, lasting three weeks. I afterwards learned that he had been subject to such attacks every winter and in cold weather at other seasons. Apparently the operative interference had released latent infection in the tonsillar crypts and adjoining lymphatics with metastatic evidences in the joints mentioned.

NERVOUS PHENOMENA.

Pfingst mentions a case of hemiplegia which did not disappear till four months after operation, also one in which there were repeated attacks of hysterical strangulation and spasmodic flexing of the thighs (Bergh). Several months ago I saw in consultation an infant, fifteen months of age, from whose epipharynx lymphoid tissue had been curetted under chloroform anesthesia, for the relief of mouth breathing, the mother said. As an undoubted result of this unnecessary surgery, every afternoon since the operation this child had had violent attacks of spasmodic laryngitis, with complete arrest

of respiration for as long as fifteen and even twenty seconds at a time, and all the cyanotic and cardiac disturbance which usually accompanies such seizures. The child had a negative history, had neved been ill and had parents of no taint whatever. Evidently the violence done her nervous equilibrium was great and causative of the attacks.

The rather severe edema of the uvula and soft palate following an enucleation of the tonsils in a neurotic woman, twenty-five years of age, had begun to disappear when I was sent for in great haste on the fifth day, as she was unable to speak, even in a whisper. The cords appeared normal and moved freely on attempted phonation, and no cause other than hysteria seemed to me likely at the bottom of the aphonia. I tried to betray her into speaking by catching her off her guard, but knowing that she was very desirous to go to Europe in a few days, I told her mother in her presence that her voice would return unimpaired by the second day at sea. This was sufficient to secure a cure, as promptly the second day, on getting up, her voice was found to be full and normal.

INJURY TO PILLARS AND ADNEXA; CONTRACTURES; FUNCTIONAL DISTURBANCES.

Laceration of the pillars is a frequent occurrence where they are friable from disease, or where much force, or rather misdirected force is applied, and will materially increase hemorrhage, especially when the posterior pillar is the part damaged. Generally these wounds heal readily with no ill effects other than prolonged convalescence, but I had the misfortune several years ago to buttonhole an anterior pillar with the punch, and the wound did not close. Moreover, the cicatricial contractures were extensive, and the two pillars adhered together on each side, limiting the mobility of the velum in such a way as to render the patient's voice nasal from time to time, though not continuously. Since abandoning the use of the punch some three years ago I have had no such perforations or contractures. While I do not seek to escape the blame for my poor technic that brought about such a result, I find myself in accord with Sewell, who says that many of these bad cosmetic and functional results are due to the faulty administration of the anesthetic. It is essential for good results in throat surgery that the patient should be kept quiet, the mouth free of

blood and mucus and the anesthetic apparatus out of the operator's way. My case bears out his theory, as the three conditions desired were conspicuously lacking.

I was rather surprised to read that Pfingst found it not unusual to buttonhole the pillars in the first twenty-five or thirty cases he had to report, and the uvula was at times removed along with the tonsil. This I have done once with the snare, and I have seen two eminent laryngologists do the same thing in this city. If the snare loop is made so small that the loosened tonsil can be just pulled through with slight force, it will be impossible to commit this surgical error, which I have likewise done once with the punch, as the parts were obscured with blood and the patient not well anesthetized. In all, I have had four cases in which the scar formation was marked, all due to indiscreet use of the tonsil punch.

Sewell reports one case of adhesion of the velum to the pharyngeal wall, shutting off one naris from the oral cavity. I had a similar case in which the velum was adherent to the pharynx on both sides, leaving an opening in the midline which scarcely admitted my little finger. As he was a mental defective, an accurate history was not obtainable, other than it was the result of a throat operation.

Where a tonsil is badly degenerated and the pillars share in the process, though the tonsil be removed with the utmost care and circumspection, the rapid formation of granulations requires removal for weeks following convalescence, if deformities are to be prevented. Such has been my experience with tonsils, the seat of infection spreading into peritonsillar abscess, and requiring removal as a prevention of recurrent attacks, though I try to prevent this new growth by local applications to the healing surface. One case, that of a woman of forty-five years of age, required free punching out of large organized granulations three times in the course of three months.

The examination of a young man's throat for the relief of pain and offensive accumulations revealed the absence of a uvula and the presence of tonsil remnants on each side, though he had had a general practitioner remove the tonsils once, and again a specialist had tried to do it, with the result already stated. Moreover, the free edges were the seat of chronic inflammation, their surfaces ulcerated and covered with a pink-

ish and yellow material, and a glistening, tense white band of fibrous tissue bounded the free edge of the pillars on one side, up and across the palate and down the edge of the opposite pillars, holding the velum in a tight grip. As the patient was pale, weak and dispirited, I would give no prognosis till I had a report from the pathologist who examined a section from each tonsil, with a negative result. As there was no glandular involvement or temperature, the tonsil base on each side was enucleated, revealing a sclerotic mass the size of an English walnut—true tonsil tissue. His voice is occasionally nasal, though the contracture is better than before my operation.

A clean removal of diseased or enlarged tonsils will at times increase the register of a singer's voice as much as two tones, but, on the other hand, it is unwise in the extreme to operate on the throat of a singer without first warning the patient what may be the outcome as to limitation of tone production. However carefully the removal be done, there will sometimes be a restraint placed on the palatal musculature by scar formation and adhesion of parts formerly free, such as to impair tone production and resonance. Such cases are on record; in fact, Stucky refers to one in his report.

Hudson Makuen goes into this particular point thoroughly and vigorously, laying stress on the carelessness with which the soft parts are handled. His explanation of why adhesions and fibrous formations alter the voice is particularly lucid, and I speak only in corroboration, since nothing can be added to his work save further evidence.

Whether we regard the enucleation of tonsils as an operation free from peril, or distinguished by an occasional mishap, at least the conviction forces itself upon us that it should not be entrusted to any but those experienced in its performance, and that the convalescence should be zealously followed. Moreover, it is essentially a hospital operation, to be done in a hospital or under the same rigid condition of asepsis and trained nursing.

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LXIII.

DECAPITATION OF THE FAUCIAL TONSIL FOLLOWED BY DIGITAL EVISCERATION OF ITS CAPSULE.*

By J. A. Ellegood, M. D.,

Wilmington.

For nearly three years it has been the almost invariable practice of the writer to eviscerate the tonsil capsule, after decapitation, when operating for the removal of the faucial tonsils in children under puberty. In nearly all of the cases the evisceration was performed by means of the index finger.

The patient is prepared as for a tonsillectomy. A general anesthetic is administered. A digital examination of the tonsils is made to determine the consistence of the tissues of which they are composed. If sufficiently soft, as it is in most cases, the hypertrophied mass is pressed through the ring of a Mackenzie tonsillotome of suitable size and the decapitation performed. In the pedunculated tonsils preliminary dissection is often unnecessary. In the case of imbedded tonsils it is generally necessary to separate the tonsil from its attachment to the faucial pillars before it can be made to protrude through the fenestra of the guillotine. The separation can frequently be performed by the index finger, the tip of which can usually be pressed up under the margo supratonsillaris into the supratonsillar fossa and brought down successively between the anterior and posterior pillars and the tonsil. During this procedure much assistance can be rendered by supporting the region of operation externally by the hand of the operator or an assistant.

In case the separation cannot be satisfactorily done by the finger, the tonsil may be grasped with forceps and instrumental means made use of for its accomplishment.

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*Candidate's thesis, American Laryngological, Rhinological and Otological Society, May 9, 1913.

In the ideal performance of the decapitation the incision should be at the junction of, or slightly inward to, the point at which the capsule and the mucous membrane meet as the later is reflected over the tonsil. Such an incision avoids cutting the strong fibrous membrane, a part of the amygdaloepiglottic fold, which lines the whole tonsil fossa and lies in front of the loose cellular tissue between it and the superior constrictor muscle.

The extent to which the tonsil extends through the ring of the guillotine can, in a great measure, be determined by the pressure of the surgeon's finger externally, at a point corresponding to the interval between the angle of the lower jaw, the anterior border of the sternomastoid muscle, and the great cornu of the hyoid bone.

Should the pressure be made by an assistant and be excessive, the degree of protrusion can be regulated by the operator in applying the instrument.

In a small proportion of cases of pedunculated tonsil the entire tonsil in its capsule will be removed by the guillotine. In the majority of cases, however, the base of the tonsil and its capsular bed will be left in situ. The index finger, armed with a stout nail, previously hardened with absolute alcohol, is introduced and the remains of the tonsil tissue broken down and scraped out. This is usually easily accomplished. Ordinarily no great amount of force is necessary to so crush or pulpify the tonsil tissue that it may be easily curetted from the capsule by the finger nail. Any that may remain attached to the edges of the capsule can readily be removed by tongue or other suitable forceps, or by a small Ballenger ecraseur.

The crushing of the tonsil tissue may be aided by external counterpressure, or the mass in the capsule can be easily pulled forward by the finger over the angle of the lower jaw, which can be used as a background.

An examination of the decapitated part will enable the operator to determine the consistence of the remaining stump. Should that, as occasionally happens, be too tough for complete removal by digital manipulation, it can be removed by such other means as the operator may elect to adopt. As a matter of experiment the lymphoid tissue in stumps of tough consistence has been removed, leaving a considerable portion of the fibrous tissue. In all such cases the remaining tissue

soon atrophied, demonstrating that the presence of lymphoid tissue is in most cases necessary to excite hypertrophy of the fibrous elements.

Not infrequently the lymphoid tissue is divided into two or three distinct masses or lobes by more pronounced septa of connective tissue which extend from the inner surface of the capsule. These should be broken down and the cavities converted into one.

It is important to remove all the lymphoid tissue. Shreds of connective tissue and the ends of the vessels which remain, speedily disappear.

If in making the section the incision does not involve the greatest diameter of the investing membrane, it should be torn or cut at its lowest part to permit of easy drainage. It can usually be easily torn with the finger.

The crypts are doubtless the cardinal features, both as regards the function and the pathology of the faucial tonsil. Of the former nothing definite is known. Normally the tonsils disappear after puberty. Although they may have some useful function to perform before this period, it has been unquestionably demonstrated that their absence by complete removal in no appreciable sense prejudices or endangers life or health. As yet no bodies have been discovered which bear the same relationship to the tonsil that the parathyroids do to the thyroid gland. It is possible that such may exist, or that some of the other lymphoid tissue constituting the "ring of Waldeyer" may vicariously, or by substitution, perform whatever part the tonsil is intended to accomplish in the process of metabolism. The crypts of largest size and greatest depth are, as a rule, found in the middle part of the tonsil, and this point is the place most desirable for beginning the process of digital evisceration.

While some of the crypts open into the supratonsillar space, none exist in any of the lymphoid tissue composing "Waldeyer's ring" outside the tonsil capsule.

The advantages claimed for the operation herein described are that in most cases it is easily performed. It is also quickly performed, thus lessening the danger of profound and prolonged anesthesia. Less assistance is required, as mopping of blood and mucus is unnecessary. The patient's head can be held in such position as to prevent the inspiration of either

of these substances. No inspection being necessary, they do not interfere with the performance of the operation. The danger from bleeding is lessened. There is no injury to the faucial pillars, nor to the venous plexus at the base of the sinus, the wounding of which is the most common cause of persistent and alarming hemorrhage. It is obvious that the more peripheral the incision the smaller the vessels cut, and the less serious the hemorrhage.

The vessels and fibrous septa are too tough and resistant to be entirely removed by so dull a curette, and, stripped of lymphoid tissue, the former readily retract and curl upon themselves so as to close their lumen. The danger from sepsis is less than when the capsule is completely removed, because the wound is less extensive and deep, and has as its background a tough and resistant fibrous membrane, instead of loose cellular and muscular tissue. In a number of instances in which the tonsils filled the entire fossa they were so covered by the plicæ that there appeared to be no peritonsillar space, and there being little or no projection beyond the faucial pillars, a section of the mucous membrane was removed from the buccal surface of the gland and evisceration digitally performed. The incubating foci represented by the crypts being eradicated, and the mucous membrane and capsule being torn below so as to afford drainage and avoid the formation of a pocket, the result was all that could be desired.

The upper portion of the capsule is surrounded by a loose areolar tissue space—the peritonsillar space. There is only a potential space round the lower portion of the capsule, for the muscular fibers of the palatoglossus and the palatopharyngeus form a close investment to it. These fibers are interlaced with one another and with those of the superior constrictor, and some of the latter are directly inserted into the capsule itself. Consequently this intimate relationship of muscle with the lower part of the capsule prevents tracking downward of infection when the lower part of the capsule and its attachment are not disturbed, whereas, such might easily occur when the lower part of the capsule is removed. The great resistance of the capsule to infection is well illustrated in gross tubercular lesions of the tonsil. In tonsils which have undergone acute inflammatory changes the capsule remains unaffected. So it apparently does after digital evisceration. Lastly, there is no

mutilation of the palate muscles, nor such injury to the mucous membrane as to interfere with deglutition, audition, or vocalization.

It may be said that such injuries are not done when total extirpation of the capsule is properly performed. While that is undoubtedly true, nevertheless, it is a well known fact that they are frequently done by the inexperienced, and occasionally by the most expert operators. It may also be said that the force necessary to perform the operation described is liable to cause serious injury to the surrounding parts, and that the remaining shreds of tissue in the eviscerated capsule are liable to lead to general infection. In the experience of the writer there has been, as a rule, but moderate reaction, the detritus has rapidly disappeared, and the denuded surface was speedily covered by extension of the mucous membrane. The insult to the tissues is by no means so great as in the old operation of Celsus—digital enucleation—revived and practiced by some distinguished surgeons of the present day.

While in some instances the child is born with hypertrophy of the faucial tonsils, the enlargement usually begins about the second year of life, and continues until young adulthood. During this age the lymphoid elements largely predominate, and the consistence of the tonsil is such that digital evisceration can be easily accomplished in the manner described. Although this operation may not commend itself to skillful surgeons, the author is convinced that it is based on good surgical principles. The results in over three hundred cases performed by him have been eminently satisfactory. In only one case has there been any recurrence of the hypertrophy, and in that case circumstances arose during the operation which prevented its complete performance. The hypertrophied mass in this case is quite smooth, by no means large, and has given rise to no disturbance. There has been no case of alarming hemorrhage, and in but one was there violent reaction. That one was operated upon too soon after an acute attack of tonsillitis, and although there was considerable reaction, recovery was complete and the final result perfect.

Simple tonsillotomy in most cases is unsatisfactory, because it leaves portions of the crypts, some of which almost invariably extend through the entire depth of the tonsil to the cap-

sule, and these are liable to become subsequently diseased. Nevertheless, it is a time-honored procedure and, except in America, the method of operation most commonly performed. Unless some operation more efficient and more easily executed is devised, it is a procedure likely to be largely performed in the future. Tonsillectomy, as performed by the most expert operators, is a more difficult operation than the average operator will practice, and complete removal by means of a tonsillotome and punch forceps, with or without removal of the capsule, is both more difficult and dangerous than tonsillectomy.

LXIV.

FRONTAL MUCOCELE.*

By Geo. W. McCoy, A. M., M. D.,

Los Angeles.

The profession differs in the definition of a mucocele, the difference being essentially in the location of the tumor. Defined in the words of St. Clair Thomson, and representing the views of the majority of nose and throat men, a mucocele is a "distension of one or more walls of an accessory sinus and an accumulation within it of a mucous secretion."

This conception of a mucocele restricts the growth to an uncommon occurrence. Hill Hastings, in his excellent article read before the society at Los Angeles, 1911, reviewing the literature, found only forty cases.

The other conception defines the term, in the language of D. Braden Kyle, as a "simple or retention cyst common in the nose and nasopharynx and the upper portion of the larynx." He also describes the condition in the accessory sinuses.

The etiology is uncertain. Turner believes a catarrhal process obstructs the natural ostium and the mucous secretions are retained, but there are cases in which the passage is patulous. The usual cause of mucocele, according to D. Braden Kyle, Thomson, and others, is cystic dilatation of a gland of the mucous membrane, or cystic degeneration of a polyp, but may be due to catarrhal stoppage of the ostium. This is called by the recent writers the new belief, and the obstructing cause is the old idea, yet some recent writers, accepting the new etiology, still restrict mucoceles to the accessory sinuses.

As there is a greater number of myxomata and fibromyxomata in the nose than in the accessory sinuses, and as the mucous membrane of the nose and nasopharynx contains more glands and has a greater number of cyst formations than

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the mucous membrane of the accessory sinuses, it is evident that conditions are far more favorable for the development of mucoceles in the nose and nasopharynx than in the sinuses. In my own experience I have removed a few tumors from the nose and nasopharynx which, if placed in the accessory sinuses, would be considered by all mucoceles, while I have only had two cases of mucocele of the accessory sinuses, and possibly a third, now under consideration.

Restricting the term mucocele to such conditions within an accessory sinus is artificial, and is not founded upon pathologic principles, and should be discontinued. The term should include any simple or retention cyst filled with mucous secretion which may become purulent or undergo secondary changes.

The following case would be a mucocele under either definition: A young man, thirty-four years of age, weighing 204 pounds, five feet nine inches in height, and in the pink of physical condition, a baseball catcher by profession, was referred to me by Dr. Guy Cochran for a tumor over the left frontal region as large as an English walnut.

The family and personal history, except the following, has no bearing. Denies venereal and alcoholic history, excepting an occasional glass of beer. Had socalled typhoid malaria at fifteen years of age; had measles when a child; with the exception of an occasional attack of grippe, has otherwise been in perfect health.

The patient has had difficulty in breathing through his nose the mucous membrane, or cystic degeneration of a polyp, but the patient's story, Dr. Rae of Binghamton, New York, removed some cartilage and bone from his nose, without much relief. The patient was advised to go west and get into the open air. He came to California in 1904 and improved immediately, probably as the result of Dr. Rae's operation. He caught ball for the Seattle and Sacramento teams, and then went to Salt Lake City, having little or no catarrhal trouble until 1910, when headaches and a full feeling appeared over both eyes, not following or accompanied by the slightest cold nor stoppage of the nose. Under treatment by Dr. Pfouts the trouble disappeared for six or eight weeks. In January, 1911, headaches and the full pressure feeling over the left eye returned. Soon a swelling appeared over the inner third of

left frontal sinus region, about the size of end of little finger. When the tumor appeared headaches ceased, but pressure feeling was always present. Dr. Landenberger aspirated a dark greenish fluid and a small opening in the frontal sinus wall was noted. The pressure feeling left, but in a month returned with the reappearance of the swelling. He was again aspirated; the patient was then sent to Dr. Schramm, who operated from within the nose, removing the left middle turbinated body. According to the patient, three weeks after the operation the contents discharged through the nose. The tumor mass soon reappeared and again discharged itself and again reappeared.

In the spring of 1912 I found a nonfluctuating tumor of the size of an English walnut over the left frontal region. The skin over the swelling was normal in appearance, but intimately connected with the mass. No opening through the bone could be felt. The left middle turbinated body and the anterior ethmoidal cells had been removed. The right middle turbinated body was hypertrophied. There was a slight mucous discharge from the nose, but none from the sinuses. Transillumination was dark over the left frontal. Dr. Soilands' X-ray plates showed the extent of the sinus and as if slightly blurred, also the tumor mass over the anterior wall of the frontal sinus, but did not show the opening in the bone. I observed the patient until August, 1912, and at no time found any discharge from the frontal sinus region. I aspirated about an ounce of a dark brown, slightly thick, but not tenacious fluid, sterile on agar and blood serum. The opening in the anterior frontal wall could be distinctly felt. Repeated and careful examination of the eyes, ears, nose and throat revealed no symptoms except those given August 31, 1912. I operated at the Good Samaritan Hospital. An incision was made along the brow and down the side of the nose. The periosteum and the soft parts were carefully elevated and retracted. An opening at inner third of the anterior frontal sinus wall, irregular in shape, about 8 mm. in diameter, was found. A small sequestrum was within the cavity, excessive bleeding clouded the liquid contents. The bony interior of the sinus throughout looked like the smooth whitish interior of a muscle shell without any membranous cover, nor could the sac of the mococele be made out. The lining of the reten-

tion sac of the soft part had this smooth appearance, but not so white in color, nor could a distinct sac of the mucocele be made out here. No dura was exposed. The nasal ostium was completely closed, and there was not the slightest sign of the sinus ever having had an outlet—just a small pit covered with pearly white bony lining where the passage should be. Through this I made a nasofrontal passage with difficulty, coming down by mistake upon the septum so that I had an opening into either nasal cavity. The anterior bony wall of the sinus being thin was entirely removed, the cavity packed with iodoform gauze and the wound entirely closed with horse hair sutures. The gauze was removed next day from the right side of the nose.

The recovery was uneventful, healing by first intention, and on the tenth day the patient left the hospital. There was slight diplopia for a month. Six months later I showed the patient to the Los Angeles eye, ear, nose and throat section. There is no deformity, no depression. The scar, owing to the slanting incision through the skin, cannot be seen over the brow, and down the side of the nose only as a faint line. At first I could pass a probe from either nostril up into the cavity. Ten days ago I attempted it and did not succeed.

Dr. Lund, who was present, advised letting the external wound heal by packing from without, avoiding making a new passage to the nose, not disturbing the mucous membrane of the nose and lessening the danger of discharge from sinus into nose. To me it seemed more or less deformity would result; the interior of the sinus I feared was too smooth to granulate, and as aspiration had been done three times, I thought the nasal drainage method the best. The patient is well. I cannot reconcile his statement of the tumor mass emptying itself twice through the nose, unless the passage was later closed.

LXV.

REPORT OF A CASE OF COMPOUND FOLLICULAR ODONTOMA OF THE SUPERIOR MAXILLA.*

By L. W. Dean, M. D.,

Iowa City.

Miss C., age twenty years, presented herself for examination on March 6, 1912. The following history was given me by Dr. Bowie, who referred the patient to me. He reported that the patient presented herself to him several weeks before, complaining of pain in the region of the upper right third molar. The third molar was sensitive. Treatment of the tooth with removal of the nerves from the root canals did not relieve the pain, and two or three weeks before I saw the patient the tooth was extracted. The tooth had the usual short, conical root. It was two weeks after the tooth was extracted that a discharge of seropurulent matter made its appearance in the socket of the tooth, and a number of rudimentary teeth were discharged through this opening.

Examination showed that there was a sinus leading upward from the root socket of the right third upper molar. On probing this sinus it was found to open into a large cavity. The diagnosis of odontoma was made and operation advised.

The opening was made into the superior maxilla, just above the gingival border, over the socket occupied by the third molar tooth. A large cavity was found which extended forwards over and external to the first and second molars. The cyst extended into the hard palate one-half the distance to the median suture. Within the cavity were found a number of rudimentary teeth and irregular pieces of bone tissue. In all, preceding the operation and at time of operation, thirty-five pieces were removed. The antrum was opened and found

*Read before the Chicago Laryngological and Otological Society, April 22, 1913.

normal. The wound was drained through the antrum into the nose and the external opening closed. Later the wound reopened and was closed by a second operation.

In connection with dentigerous cysts and empyemas of the antrum of Highmore, with extensive necrosis and the operation for these conditions, I have been experimenting with various methods for closing the wound between the cavities and the mouth. The method that I have found most successful was to remove the bone from the alveolar process so as to get good apposition of the periosteum covering the external and internal surfaces of this process.

I found that if the wound were closed with silk sutures they would cut through and the wound reopen. Catgut was next tried with similar result. Our best results have been secured by using silkworm gut and placing along each side of the alveolar process a small piece of very narrow rubber tubing. Over the tubing, on the inner surface of the process, the silkworm gut is looped, the ends being tied over the tubing on the external side of the process. In all, during the last eight months, I have operated on some fifteen cases. The last seven or eight have been operated upon by this method with uniform good results.

It might not be amiss here to quote from the most recent authorities in regard to odontomata. It is evident that but little is positively known except as to clinical experience and treatment. The etiology is still within the realm of speculation. There are several kinds of odontomata, or tumors composed of one or more of the dental tissues arising either from teeth changes, or teeth in process of development.

Sutton classifies the different forms of odontomata as follows:

Epithelial Odontomata.—These develop from the remains of the epithelium of the original enamel organ. They appear in the form of a series of cysts separated by thin septa and contain mucoid fluid.

Follicular Odontomata or Dentigerous Cysts.—They usually form in connection with developing permanent teeth. The inner wall of the cyst is the remains of the dental follicle, within which, in every instance, there is a tooth or remains of a tooth. The cyst is filled with fluid, usually serous or mucoid.

I believe this fluid always contains cholesterin crystals. The contents are not purulent unless infection comes from without. The walls of the cyst are bony. They may become so large as to bulge out and cause great deformity.

Fibrous odontomata are developed from the connective tissue elements of the developing teeth. These are usually seen in animals.

Cementomata, the result of cement forming around the teeth, are rare in man.

Radicular odontomata form after development of crown and while root is forming.

Composite Odontomata.—These are abnormal growths of all the elements of tooth structure, resulting in solid, illformed masses of cementum, dentine, and enamel.

Compound Follicular Odontomata.—Socalled because it results from several tooth follicles. The follicle wall becomes thickened into a fibrous capsule. In this there may be portions of dentine, enamel, or cementum, imperfectly or more or less perfectly formed teeth, all together in a heterogeneous mass. Rudimentary or other teeth have sometimes been found in very large numbers; as many as several hundred have been taken from the jaws of one person. (Oral Diseases and Malformations, Brown.)

These tumors have been observed in the human subject, and in goats, horses, and other animals. In man they usually develop in early life, during the period of second dentition. Among the records of cases of this character the youngest was a boy ten years old, and the eldest a woman of twenty-seven years.

The number of denticles found in tumors of this character varies greatly. From three to four to as many hundreds have been removed from the jaws of a single patient.

Among the recorded cases of especial interest is one which will be briefly mentioned:

Tellander reported a case of tumor of the right side of the superior maxilla in a woman twenty-seven years of age, which had been growing since she was twelve years old. The character of this growth was a hard, painless swelling. It occupied the space in which should have been lodged the first molar, the bicuspids and the cuspid of the permanent set. These

teeth had not erupted. Upon opening the tumor it was found to contain several minute teeth; nine individual teeth with conical crowns and conical roots, each complete in itself, the crown being furnished with enamel, and six masses of dental tissue having the appearance of being formed by a union of several single teeth, and all presenting the characteristics of supernumerary teeth. Another tooth made its appearance about a year later in the location from which the tumor was removed. (Ottolengui, Items of Interest, February, 1913.)

LXVI.

CASE OF MENINGITIS IN WHICH THE CISTERNA MAGNA WAS DRAINED.*

By Norval H. Pierce, M. D.,

Chicago.

O. L., aged ten years, schoolboy.

Diagnosis.—Mastoiditis; purulent meningitis.

History.—Patient had had a chronic suppurative otitis media for nearly two years.

Present Condition.—Temperature on admission was 101.6.° Some rigidity of neck. Kernig present. Babinski. Pupils not contracted and eye grounds apparently normal, excepting that the veins appeared just a trifle large.

Patient was admitted to the hospital on March 10, 1913. The following day the leucocyte count was 7,500. Lumbar puncture was made, and fluid came out with considerable pressure and was cloudy. On March 12th, examination by Dr. Pierce showed spontaneous nystagmus on both sides. Heard a^1 fork. No symptoms of fistula. Radical mastoid operation was performed by Dr. Pierce on that day, and necrosed bone found in roof of tympanum and posterior wall of mastoid, extending back along the skull. Black necrosed bone removed and dura exposed. The next day the patient was no better—all the symptoms of meningitis were the same. Lumbar puncture: ten cubic centimeters, under pressure, cloudy. Specimen sent to Michael Reese Hospital, where cultures were made and pneumococcus in pure culture reported present; one hundred and sixty cells to field, polymorphonuclear predominating; Noguchi plus.

March 14th.—Lumbar puncture—twenty cubic centimeters. Marked pressure; cloudy. Stiffness of neck more marked. Kernig marked. Babinski. Patient cried out constantly and complained of severe headache.

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*Read before the Chicago Laryngological and Otological Society, April 22, 1913.

March 15th.—Drainage of cisterna magna by Dr. Pierce. A trephine opening was made one inch below external occipital protuberance and the opening enlarged with rongeur forceps. No pulsation present. Dura and arachnoid opened and considerable cloudy fluid allowed to flow out, and immediately pulsation was seen. A gutta percha drain was inserted and two pieces of xeroform gauze inserted external to dura, one above and one below gutta percha drain, and the wound closed. Voluminous boric and alcohol dressing applied. Blood pressure taken during the operation fluctuated between 110 and 95.

March 16th.—Patient's temperature went down after the operation for several hours, but again up to 101° on evening of the 16th. Leucocyte count, 3,000.

March 17th.—Wound dressed twice a day. Drainage. Blood pressure went down to 75. Patient's limbs and abdomen bandaged and strychnin given. Blood pressure went up to 95.

March 18th.—Temperature, 101° to 103.° Wound dressed twice daily and draining slightly. Blood pressure 90. Patient cried out often and complained of some headache. Emaciation marked.

March 19th.—Temperature, 100° to 102.° Headache, and patient cried out. Drain out of wound. Wound reopened by Dr. Pierce, allowing considerable cloudy fluid to escape and rubber drain reinserted. Blood pressure down to 80.

March 20th.—After reinserting drain, temperature from normal to 99° for twelve hours, after which it again went to 101.°

March 21st.—Patient was getting stupid and showed evidences of marked sepsis. Temperature, 101° to 102.° Blood pressure 90. Involuntary stool and urination at times. Patient not unconscious, but slept a great deal, but perfectly rational when aroused. Wound was draining, but looked dull and septic.

March 22nd.—Patient much worse; sepsis more marked; marked spontaneous nystagmus; stuporous, but could be roused.

March 28th.—Patient died.

We have here a case of undoubted septic meningitis. All the classic symptoms are present—stiff neck, engorged veins in the retina, Babinski, Kernig, headache, spinal fluid showing a large percentage of white and polymorphonuclear corpuscles and a virile microorganism. It is interesting to note

that the germ which was grown from a spinal puncture apparently differed from the germ which was procured directly from the cisterna magna. However, I believe that this is a bacteriologic error, as the germ which was taken from the cisterna magna resembled very closely but was not the pneumococcus, and differed from it principally in that it was hemolytic.

We depend for our diagnosis on lumbar puncture in meningeal inflammations. Still, the findings are not always useful in differentiating between serous meningitis, localized meningitis which is permanently circumscribed, diffuse meningitis, or one that is spreading. In all probability, if we can recover a live pathogenic bacterium from the spinal fluid, we have to do with meningitis which is already diffuse or is at the time spreading. Other than this, we may have an enormous cytologic content in either a serous meningitis or a meningitis which is permanently circumscribed.

It is very important, in contemplating the performance of this operation—draining the cisterna magna—to differentiate between a serous meningitis, a circumscribed meningitis, and a diffuse meningitis, for I do not believe that the operation is indicated in anything but a diffuse meningitis, or a meningitis which tends at the time to spread. The two former conditions —serous meningitis or circumscribed meningitis—may best be treated, I believe, by spinal puncture or permanent lumbar drainage. If the base of the skull is bathed in pus—in other words, if the cisterna is an abscess cavity—then this operation is indicated. I can readily imagine how the unavoidable trauma incident to the operation of draining the cisterna magna, the extreme and sudden depletion of the subarachnoid space of its natural fluid, might transform a circumscribed meningitis into a diffuse meningitis. The fact that it has the advantage over splitting the dura in the mastoid wound (a septic field) is to a certain extent true, for in our case the external incision healed by first intention, and when the drain had to be replaced it was found that the dura itself had largely healed; it cannot be considered as aseptic or as free from the danger of infection as, for instance, a surgical wound made in the skull in the course of an operation for tumor, for in our case the septic wound in the mastoid is only a short distance from the wound in the occiput, and both are covered practically by the same dressings.

How, then, will we decide as to when is the proper time for

opening the cisterna magna—for this operation has a place in surgery. If the headache is severe, temperature high and stiff neck increasing, we should depend first on lumbar puncture. The cytologic, bacteriologic and chemical condition of the cerebrospinal fluid should be carefully studied. In the absence of living bacteria we should continue with our lumbar drainage. If, however, the cytologic content increases and the clinical picture is that of an increasing meningitis and we recover a living pathologic bacterium from the fluid, we may resort to opening the cisterna magna. However, I would raise my voice in warning against participating in a hope which, in my opinion, will be vain.

There are two factors with which we have to deal in a meningeal inflammation: First, and least important, the increase of the cerebrospinal fluid. Second, the inflammatory process which takes place in the pia. It is the latter process which kills, and we may drain off the cerebrospinal fluid for days, and even weeks—as we did in our case—and the patient succumb to the meningitis. It is the inflammation about the sheaths of the blood vessels which is carried in the pia to the vital center of the brain and medulla which kills. I doubt if subdural drainage of any kind will influence the disastrous process in the pia, once it has become diffuse.

The operation itself is easy of performance. The technic has been thoroughly described by Dr. Haines. I believe, however, it is right to caution against the too sudden release of the fluid, which is usually under great tension, from the cisterna magna. This may best be obviated by introducing a needle through the dura and arachnoid after the bone has been removed. It will take some time, of course, before sufficient fluid has been removed in this way, before the pressure has been reduced to any considerable extent, but there is no reason why a very small capillary tube should not be inserted into a very small opening at the first sitting and then, after another subsequent dressing—say within the next twenty-four or forty-eight hours—the dura can be fully incised, if necessary, and examination of the foramen of Magendie made.

The fall in the blood pressure is a very dangerous sign. It may go very low, however, without terminating fatally. We should be ready at the time of operation to bind the extremities and the abdomen in case of syncope, and afterwards, in the course of the disease, when the blood pressure gets below 100, the same procedure should be carried out.

LXVII.

THE USE OF VACCINES AFTER THE MASTOID OPERATION.

By William C. Braislin, M. D.,

Brooklyn.

I have employed vaccines in six cases, subsequent to the mastoid operation, out of a total of thirty-five mastoid operations performed during the past winter. In the ordinary, smoothly convalescing cases they have not been used. They were used in cases presenting the following complications:

1. Extensive infiltration of the glands of the neck following an operation for streptococcic mastoiditis and cervical abscess.
2. Indeterminate or obscure causes of elevated temperature (the lateral sinus excluded from involvement).
3. Delay in granulation and healing processes in mastoid wound.
4. Pain in limbs, joints and back, without special elevation of temperature, after an operation for streptococcic mastoiditis.
5. In a case of postoperative pneumonia.
6. In a case of infection of the labyrinth.

Case 1.—Streptococcic infection of right mastoid in a little girl of two and a half years. The mastoiditis was complicated in this case by a very extensive abscess on the corresponding side of the neck, which was operated upon at the time of the mastoid operation and a drainage tube introduced into the depth of the wound. The cervical abscess was deeply seated and surrounded by enlarged lymphatic glands, one of which protruded into the right lateral and posterior wall of the pharynx. A culture taken at this time and submitted to Dr. Archibald Murray showed the infection to be streptococcic. The child did well for three days after the operation, but on the following day the lymphatic glands of the neck on the opposite side became tense, markedly swollen and tender, espe-

cially at the angle of the jaw, and a distinct rise of temperature occurred. No involvement of the mastoid was discoverable on this (the left) side. The culture made at the time of the operation not being available for making an autogenous vaccine, we began the use of a stock (antistreptococcic) vaccine, a minim being given daily for four days, then on alternate days for four days more. The glandular swelling had begun to decline within twenty-four hours of beginning treatment. The discharge of pus from the wound lessened rapidly likewise. The temperature became gradually normal, and there appeared to be a sensible increase in the rapidity of the healing of the mastoid wound. The neck wound healed within three weeks from the time of operation; the mastoid wound, at the end of the sixth week.

Case 2.—Pneumococcus infection of both mastoids, ten days after eruption of measles, in a girl three years old. Both drums were promptly incised. Cultures taken from both ears were reported by Dr. Murray to be pneumococcic. The discharges continued to be profuse from both ears, and the temperature continued high. The right mastoid, which was tender to pressure, was operated upon, the sinus being exposed but healthy. The antrum and cells contained much free pus. An autogenous vaccine from a culture removed from the antrum was started in course of preparation, at our request, by Dr. Murray. Much to my disappointment, the temperature did not subside to normal, but continued nearly as high as before the operation, between 102° and 104,° so that after excluding the lungs from any influence in the production of the temperature, operation was performed on the left mastoid on the third day following the operation on the right. Flaky pus was found in the antrum. We now believed that the temperature would fall to normal, but in this we were again disappointed. Some few pus cells were found in the urine at this time, but not enough to indicate a pyelonephritis. The lungs remained clear, though mucus and some blood appeared at times in the stools, indicating a condition of the colon which affected the temperature in some degree. Three days after the second operation the autogenous vaccine was ready for use, the first culture having failed to grow with sufficient activity to provide a vaccine.

Some reaction followed the inoculation of the vaccine, the

temperature even rising to 105° the second day after its use. From this point on, a rapid subsidence of the temperature took place. On April 24th, a small skin abscess of the scalp which had newly developed, with a slight rise of temperature, was incised. Both wounds are doing well.

Case 3.—A tall girl of fourteen years, large for her age, always rather delicate; has grown very rapidly for the past two years. Her mastoiditis of four days' duration followed a severe attack of tonsillitis, accompanied by profuse discharge from the ear, extreme tenderness of mastoid and high temperature.

At operation (January 10th), an extensive edema existed in the tissues over the mastoid. Blood stained serum was encountered throughout the diploetic mastoid. Free pus was found directly in contact with the lateral sinus, which was exposed externally and anteriorly for a length of three-fourths of an inch, but was apparently healthy. After the operation the temperature rose but once over 101° F., but the patient complained greatly of pain in her limbs and back. This continued to be the cause of much complaint, and on January 14th one-half cc. of an autogenous streptococcic vaccine, containing 50 million bacteria to the cc., prepared by Dr. Murray, was given. The afternoon following the first dose the temperature rose to 101.2,° with an aggravation of the pain in the limbs and back. The next day she was much more comfortable than before the inoculation, the pulse rate became slower and stronger by the second day, and on the third day the pain had left the limbs and she seemed brighter and more comfortable. Throughout her convalescence, which was rather prolonged, more or less tenderness of the joints persisted, and a mild attack of chorea occurred about the time of the complete healing of the mastoid wound. I am inclined to believe that these symptoms would have been in less degree if we had persisted in the use of the vaccine.

Case 4.—Harry S., age nine months, a delicate looking, wasted infant of mulatto (West Indian) parentage, was received at the Brooklyn Eye and Ear Hospital for operation. There was a tense swelling above and behind the right ear. A purulent discharge from the ear had existed for an indefinite period. Operation was performed November 19, 1912. On incision, much pus was liberated, besides which the swelling

contained flaky fibrinous masses of a cheesy consistency, which were curetted away. A sinus in the bone led to the antrum. The malleus and incus, which were removed, appeared necrotic. A small area of healthy looking dura was exposed above and posteriorly. Two weeks later a swelling appeared on the opposite (left) mastoid region, and on December 5th this was also operated upon by Dr. Bouvier, senior house surgeon of the hospital, who reported that thick curd-like flakes of pus similar to the condition found in the right had been likewise found in this (the left) side. Neither wound made the least progress toward healthy granulating or healing. Mercurial inunctions were prescribed, but an irregular septic temperature developed, and the child became still more emaciated. A polyvalent vaccine (Van Cott's) was administered every fourth day for a period of four weeks. The child died February 6, 1913, having been in the hospital ten weeks. Dr. Murray performed an autopsy. Both mastoid wounds were clean, but almost as little healed as when operated upon. The brain was entirely normal and uninfected. The dura was exposed over three small areas, two on the right, one on the left side. Both lungs were extensively consolidated. A pleurisy of a serofibrinous type was present, the fibrin being deposited in thick layers. A very large abscess was found in the middle of the right lung. This was the only fatal case in the series.

Case 5.—Radical mastoid for chronic suppuration, infection of the labyrinth. A boy of eleven years was operated on November 15th, for chronic suppuration of the left ear, existent since infancy. A radical mastoid operation was performed. The incus was absent. Vomiting persisted for three days after operation. No nystagmus, tongue coated. Calomel was ordered, after which the bowels moved and the nausea ceased. Two days later (21st) the temperature rose to 102° F. (mouth), and persistent vomiting recurred, especially on rising in bed. Vertigo as if falling forward (not toward the affected side) and severe headache. Slept but little. Next day more comfortable. Mind perfectly clear. Some stiffness of neck, temperature remitted, but again rose to 102° in the afternoon. Polyvalent vaccine one-half cc. was administered every four days, the temperature continuing irregular for a period of three weeks, meanwhile assuming a flatter curve

and a gradual cessation of all untoward symptoms. Ear entirely healed and dry at the end of nine weeks, but hearing nil in this ear.

Case 6.—A man, thirty-six years of age, with marked mastoid tenderness and severe pain in left ear; for six days profuse discharge from left ear, with a history of previous ear trouble. Operated February 27, 1913, the culture from mastoid antrum was reported by Dr. Murray to be pneumococcus. The next day he complained of severe pain in the right side of the chest, especially on deep breathing. Dr. Ritter, senior house surgeon, applied wide bands of adhesive plaster. The temperature on the next day (March 1st) rose to 104.4.° No consolidation could be detected. On the next day, Dr. Frank L. Tucker examined the chest at my request, and found a pneumonia developing, an area of consolidation in the right lung being clearly made out. On March 3rd, and for three days thereafter, a stock pneumovaccine was administered daily, each dose representing 40,000,000 bacteria. On March 4th the temperature came down to 99.4,° with profuse perspiration, and did not after rise above 101.° Recovery rapid and without further complications.

LXVIII.

ON THE EFFORTS OF NATURE TO CURE SEPTIC THROMBOSIS OF THE LATERAL SINUS.*

By Harry Friedenwald, M. D.,

Baltimore.

Two cases have come under my observation which impressed me with the curative efforts of nature—if we may so call the processes which tend to circumscribe and eliminate the infected thrombus in the lateral sinus. When we consider the desperate prognosis of untreated cases, we are warranted in giving our attention to these observations, together with those that have been reported hitherto. In doing so there is not the slightest intention to minimize the danger of untreated thrombosis.

Case 1 has already been reported in another connection to this society.[1] A brief statement is as follows:

In December, 1913, I was called to see a child, two years of age, who had within a few months suffered from an attack of scarlet fever, followed by measles. Double purulent otitis media was the result. A swelling had appeared over the left mastoid, which a physician had incised three weeks before I saw the patient. I found a marked hard swelling, about an inch behind the incision, over the mastoid. The ears were both discharging. On the following day the mastoid operation was performed. No visible opening into the mastoid was found in the surface of the bone, which contained pus and granulations. The mastoid process having been cleaned out, a second incision was made, one inch perteriorly, into the abscess, and much pus evacuated. The bone was denuded. The patient made a good recovery, but both wounds continued to suppurate, and excessive granulations formed. In dressing the wounds we found that a probe could be passed far into the

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posterior one, but it was not possible to determine its exact position.

On February 3rd, the patient having been admitted into the City Hospital (Baltimore), I found that the probe passed far into the skull cavity. An incision was made which joined the two previous incisions and the bone was exposed. It was then found that a fistula extended from the posterior wound into the skull, the opening being situated in the mastooccipital suture. The opening was not more than five or six mm. in diameter and perfectly circular. It was enlarged and the bone removed forwards towards the ear, thus exposing the fistulous canal. We then found that the pus had not come from an extradural abscess, but that it was the lateral sinus itself which was continuous with the fistula. A grooved director was passed into the canal and it was slit open. In the sigmoid portion the part was found where the sinus had been exposed to the mastoid disease and where the infection had occurred. It was evident that a septic thrombus had formed, but had been securely shut off from the circulation by firm clots, and that the pus had evacuated itself through the emissary vein, thus producing the abscess.

The case just described furnished an example of the limitation of the septic thrombus which is to be regarded as a conservative effort of nature.

Case 2.—Katie S., aged eight years, was admitted to the Mercy Hospital (Baltimore), November 20, 1912. She had had measles when two years old, but not scarlet fever. The right ear had been discharging since she was five years old, the discharge having a bad odor for some time. When the discharge was free there was no pain, but when the ear was not running she complained of great pain in the head. For about a year there were frequent recurrences. The pain would last a few hours and would then pass off. She was able to go to school until four days before entering the hospital. At this time she became quick sick, and a physician was called in, who thought she had typhoid. She had high fever, was delirious and very weak.

On entering the hospital we found destruction of the right tympanic membrane and the middle ear filled with foul pus. There was no marked sensitiveness over the mastoid or over the course of the jugular vein. Her temperature showed a

pyemic character, with mild chills, high rises and rapid falls of temperature, as follows:

November 20th, 4 p. m., 100.4°; 8 p. m., 100.8°; 12 m., 105.4°.

November 21st, 4 a. m., 103°; 8 a. m., 102.4°; 12 n., 98.8°; 4 p. m., 98°; 8 p. m., 99°; 12 m., —.

November 22nd, 4 a. m., 99.6°; 8 a. m., 97.6°; 12 n., 97.8°; 4 p. m., 97.8°; 8 p. m., 98°; 12 m., 101.8°.

November 23rd, 4 a. m., 99°; 8 a. m., 98°; 12 n., 97°; 4 p. m., 97.8°; 8 p. m., 101.8°; 12 m., —.

November 24th, 4 a. m., 102°; 8 a. m., 100.4°; 12 n., 98.2°.

The urine showed some albumin and a few hyalin and granular casts and pus cells. The blood examination revealed polynuclear 76 per cent, small mononuclear 17 per cent, large mononuclear 6 per cent, and a total blood count of 20,500. A blood culture made on November 23rd, on agar plate, bouillon and serum water, was sterile. The eyegrounds were normal.

The operation was performed November 24, 1912. Ether anesthesia. The regular mastoid incision was made, extending from the level of the top of auricle down to a point below the tip of the mastoid. The bone was exposed widely on both sides. A minute fistulous opening was found above the mastoid antrum, from which pus of a very foul odor exuded. This opening was enlarged with chisels; the bone was very sclerotic; a large cavity was entered, filled with the same character of pus, pulsating actively. The sinus was found exposed extensively. The antrum was then opened and contained a cholesteatoma. A radical mastoid operation was performed, thoroughly cleaning the middle ear of pus and cholesteatomatous masses. The bone over the sinus was next removed upward to the knee and downward to the bulb. The upper half of the sigmoid sinus appeared distended and covered with coarse, thick granulations. The lower half was smooth, whitish and seemed collapsed. On close examination the entire wall of this part of the sinus was seen to have been destroyed over a distance of about 2 cm. Venous blood flowed freely from the upper and after-incision. A probe was introduced and, when followed down a short distance, showed firm closure of the sinus and demonstrated the complete destruction of outer wall below. Bleeding was stopped with pressure. At the lower end an opening was found entering

the sinus for a short distance and discharging pus. For evident reasons the jugular was not tied. A plastic was made by splitting the posterior part of the canal lengthwise, with incisions downward and upward in the form of a "Y." The canal was packed and a drain placed into the lower end of the sinus, and the wound was dressed as usual. From the pus taken from the mastoid wound, short chain streptococci developed in pure culture.

The temperature after the operation became normal and at no time rose to 100.° The patient made an uneventful recovery and was discharged from the hospital January 2, 1913, the wound almost completely healed.

In this case it is seen that the sinus was completely and firmly closed at both ends, and that the thrombosed portion had relieved itself entirely by destruction of almost the entire outer wall of the affected portion.

These cases brought to my mind others in which complete closure was found at one end of the thrombosed sinus—or even at both ends; in the latter case so firm as to convert the diseased part of the sinus into a tensely filled pus sac.

A number of cases have been reported in which complete obliteration of the sinus by collapse or, more probably, by organized thrombus has been observed, either during the operation or after death. Thus Griesinger[2] described a post-mortem case of complete obliteration of the entire sigmoid sinus by a firm thrombus. Schwartze[3] published a like case in 1873. Similar cases were also published by Alt,[4] Politzer,[5] and Zaufal.[6] Török[7] reported a case in which there was injury of the sinus during an operation. Six months later, pyemic symptoms; the sinus was exposed and found to contain, from the tip of the mastoid to the knee, an organized thrombus of firm connective tissue. At the autopsy the obliteration was found to extend to the torcular. Purulent phlebitis had developed beyond this and had been the cause of death. Grüning[8] found destruction of the wall of the sigmoid sinus and a perisinus abscess and the peripheral portion of the sigmoid sinus converted into a solid cord. Barr,[9] in a case of temporosphenoidal abscess, found "complete occlusion of the sigmoid sinus, the walls of which were converted into a fibrous cord." Suckstorff and Henrici[10] report a case of spontaneous cure of thrombosis; at the autopsy the transveres sinus was

found to be completely obliterated by connective tissue. The thrombosis had run a latent course.

A number of cases are reported of thrombosis running a nonfebrile course in which obliteration of the sinus took place.[11]

A number of these cases presented fistulous openings in the sinus walls. The nonfebrile course is variously explained—by the aseptic character of the thrombus, by the mild nature of the infection or by the discharge of pus when formed through an opening in the external wall.

We cannot determine with certainty how the obliteration takes place—whether by direct organization of the thrombus without its disintegrating, by emptying of the pus formed through the outer wall, or by collapse and agglutination of the walls.

A large number of cases bearing marked similarity to those reported by me are to be found in our literature. Hoffmann[12] reports one case in which the central and peripheral ends of the thrombosed sigmoid sinus at the operation were found to be closed by connective tissue obliteration of the sigmoid sinus[13] in a child with extensive defect of the inner wall of the mastoid process and extradural abscess. On splitting the sinus, it was found firmly closed by organized thrombus with but a little of the lumen retained. The fever subsided immediately. Wernicke[14] found the sigmoid converted into a 2.5 cm. long and 1.0 cm. wide abscess, completely shut off above and below by organized masses. In a second case the obliteration existed only toward the central end. These cases were cured by operation. Wernicke gives a complete bibliography of the early cases up to 1900. Streit[15] reports a case of spontaneous cure of thrombosis by obliteration of sinus. There were no symptoms of sinus thrombosis. The operation was done for mastoiditis and exposed a cavity whose inner wall was covered with granulations and was found to be the obliterated sinus. Hölscher[16] reports one case in which all symptoms of thrombosis were absent, while in the second there were rigors and swelling in the neighborhood of the emissary veins, but otherwise only headaches and slight evening temperature. On opening, a perisinus abscess was found; the sigmoid sinus was absent in both cases, being replaced by granulation tissue which passed over directly posteriorly into

the normal sinus. He reports a third case of death from cerebellar abscess without symptoms of sinus thrombosis; no sinus was found at the operation. At the postmortem the transverse sinus was found enlarged from torcular to knee, and from this point on completely closed by a firm thrombus. Bárány[17] describes a case of spontaneous cure of sinus and bulb thrombosis. The case was one of mastoiditis with extensive subperiosteal abscess. On opening the mastoid the sinus and dura were found extensively exposed, greatly thickened, and in the neighborhood of the bulb a black opening was found in the sinus through which a sound could be entered for a short distance in both directions. The sinus was thin and obliterated. Recovery took place without further operation. In the discussion Urbantschitsch[18] stated that he had demonstrated a similar case of cured sinus thrombosis with obliteration of the lumen. On exposing the sinus in a case of putrid cholesteatoma, a tablespoon of foul green pus was discharged, the wall of the sinus, which was discolored, was slit up; the whole sinus was found filled with nonpurulent thrombotic masses. Toward the sinus transversus the sinus walls were completely adherent to each other.

Recent experimental studies of this subject have brought interesting results. Haymann[19] was able to produce and study sinus thrombosis and pyemia in dogs and monkeys. Among his conclusions the following are to be specially mentioned: The thrombi in their earliest stages may contain microorganisms, or the latter may enter the thrombi subsequently from without or be brought to the thrombi by the blood stream. The number of organisms contained in the thrombus vary greatly, but the ends of the thrombus are usually most benign. A moderate number of organisms does not prevent organization of the thrombus. Experimental infected thrombosis shows a marked tendency to spontaneous recovery; and even malignant forms, in which the only wall was destroyed, at times showed organization, vascularization and recanalization of the sinus.

In a subsequent paper Haymann[20] considers the processes tending to spontaneous cure of sinus thrombosis in man. He reports a case of purulent otitis media for a year with death from meningitis. The sigmoid sinus was obliterated throughout a large part, completely closed by firmly organized connect-

ive tissue with distinct evidences of beginning recanalization, a condition similar to his findings in experimental thrombosis. He insists that the thrombosis must not be looked upon as non-infected because of the spontaneous recovery, though the infection may not have been very virulent. This conclusion is supported by his experimental observations. This is the first case in which histologic proof is brought of complete spontaneous cure of a sinus thrombosis of otitic origin. He refers to the case of Miodowski,[21] in which there were similar histologic findings, but points out that though the jugular and sinuses were thrombosed, the infection was from a cervical gland and therefore not otitic. He concludes that processes tending to spontaneous recovery in sinus thrombosis in man are not of rare occurrence. The recovery may be so complete that recanalization is established.

Voss and Haymann have emphasized the importance of these processes in their practical bearings. Voss,[22] in a paper bearing the startling title "Lasst den Thrombus in Ruh," warns against the removal of the thrombus with the sharp spoon; he points out that the entire outer wall of the diseased part should be carefully removed, and he only incises the thrombus. The practice to leave the firm ends undisturbed would appear to be in accord with the experience which the cases described and referred to afford. A source of danger which must be emphasized is that of incising the collapsed sinus or the inner sinus wall when the outer wall has been destroyed, with the intent of opening the sinus. Thus Grünert and Dallmann[23] reported a case in which the sinus became obliterated after injury during operation, and an incision through the fibrous cord passed directly into the cerebellum. In another case, Uchermann[24] in this manner opened a cerebellar abscess by incising through a collapsed lateral sinus. And Herzfeld[25] had a similar experience in a case of cholesteatoma. He opened into a sinus, the outer wall of which was covered with granulations, and incised directly through the obliterated sinus into the brain. The patient recovered.

The firm closure of both ends of the thrombus with breaking down in the middle is less apt to lead to metatasis and general infection. But, as Hessler[26] has shown, the advantage is not entirely on this side, for those cases in which general infection occurs are less apt to develop cerebral abscess and meningitis, and vice versa.

Another, though remote, source of danger was pointed out by Hoffmann,[27] namely, that an organized thrombus may subsequently disintegrate and lead to grave results. Brieger[28] reported a case of such reinfection of a healed sinus and jugular thrombosis.

We are warranted in concluding that the course of a thrombosis varies according to the virulence of the infection and other influences. It may become organized without disintegration, and the lumen may even become reestablished. But even in those cases in which it becomes purulent in its central part, the firm closure of both ends and the formation of a fistula or the entire destruction of the outer wall may permit its more or less successful drainage, and thus tend to its spontaneous cure. This process in its nature is the same that takes place in every case in which conditions are brought about by operation which permit the formation of a noninfected thrombus at both ends of the infected portion of the sinus. It is apt to be incomplete when spontaneous, since the anatomic structure of the temporal bone places many obstacles in the way of its complete and easy drainage.

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LXIX.

BONE CONDUCTION IN SYPHILIS.*

By Dr. Oscar Beck,

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In Stuttgart, during a discussion of a paper by Voss, I made a preliminary report of my investigations concerning bone conduction in syphilis, which subject I should like to take up more in detail in this article.

During the examination of a great number of patients infected with syphilis, who at the same time suffered from disease of the ear, I observed that bone conduction was strikingly reduced in comparison to the shortening of air conduction in affections of the internal ear. It was very evident, also, that lengthening of bone conduction frequently could not be so distinctly elicited in lesions of the sound conducting apparatus as we are accustomed to obtain it in nonluetic patients troubled with a similar ear condition. Since, however, all our methods of measuring bone conduction are liable to subjective variation, and since there was the possibility of a lengthened bone conduction in the examiner (in this instance myself), I had my results controlled by other examiners, whose findings did not differ materially from mine. This shortened bone conduction in syphilitic affection of the ear is mentioned in several places, as I found in a subsequent search of the literature. But the almost regular occurrence of this symptom and its practical value in diagnosis, especially in the differential diagnosis between luetic and nonluetic diseases of the inner ear, so far have not received their proper recognition, although Wanner, during the discussion in Stuttgart, stated that for a long time, on the basis of this symptom, he had been able to diagnose syphilis.

In the first place, I deemed it necessary to make a careful study of bone conduction in syphilitics who complained of no

*Translated by E. Roth, M. D.

ear symptoms, and in whom an otoscopic examination revealed normal conditions. The method which I employed for this purpose was originally quite crude. I applied a vibrating tuning fork of middle register to the mastoid process of the patient, and when it ceased to be heard by him, I immediately transferred it to a similar position on the mastoid bone of a nonluetic person with normal hearing.

In spite of the fact that this method has many chances for error, I was able in the overwhelming majority of cases to demonstrate that differences exist between the bone conduction of normal hearing luetics and the bone conduction of normal hearing nonluetics. I examined patients with all kinds of physical manifestations of syphilis, particularly those of the second stage. Examinations were made also of patients with latent syphilis, whose history and positive Wassermann reaction proved the existence of the disease. In this series of cases were included also patients who had been infected with syphilis, but who, after various courses of treatment, gave a negative serum reaction.

The table, which was prepared at my suggestion by Dr. A. W. Hilger of St. Paul, Minnesota, may now be referred to. In this collection no cases were selected, the findings being the results obtained in fifty-six patients sent to our clinic for otologic examination according to the usual routine before the administration of salvarsan.

From this table, which was prepared two and one-half years ago, it would appear that only in a small percentage of cases is the shortened bone conduction not found—which is a fact. Since then, however, I have observed larger series of cases, and in these (as is to be expected when a large number of cases is studied) the percentages varied materially. Patients with eruptive manifestations of syphilis are found in whom normal bone conduction is elicited, and the same is true of patients with tertiary symptoms. In cerebral syphilis, which provokes symptoms of various cranial nerve involvement, the shortening is practically never absent. In the metaluetic types of the disease, such as tabes or paresis, the symptom must be cautiously used. For here we are dealing mostly with subjects on the further border of middle age, in whom, independent of the disposition to arteriosclerosis produced by syphilis, phys-

No.	Name.	Age.	Duration of Lues	Eruption	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
1	E. Z.	45	6 months	—	Normal	Never	Normal	R. 5; L. 5
2	A. J.	32	3 weeks	—	Normal		Normal	R. 8; L. 10
3	A. D.	28	3 weeks	—	Normal		Normal	R. 8; L. 13
4	K. N.	49	3 weeks	—	Normal		Normal	R. 7; L. 11
5	C. E.	30	6 weeks	+	Normal		Normal	R. 12; L. 5
6	J. H.	33	5 months	+	Atrophy L. and R.		Normal	Normal
7	D. D.	—	3 years	+	Normal		Normal	R. 17; L. 30
8	K. M.	20	6 weeks	—	Normal		Normal	Normal
9	J. N.	26	5 weeks	+	Normal	Noises	Normal	R. 8; L. 10
10	B. G.	26	7 years	+	Chr. supp. L. and R.		Shortened	Normal
11	J. P.	34	6 weeks	+	Normal		Normal	R. 14; L. 10
12	F. J.	37	5 years	+	L. scar; R. chr. adhesive processes	Noises	Normal	R. 7; L. 10
13	M. Z.	29	8 weeks	—	R. and L. cicatrices	R. and L., Discharge during childhood	Normal	Normal
14	A. N.	37	2 weeks	—	R. norm.; L. O. M. S. C.	L., Discharge	R., Norm.; L., Short	R. 4; L. norm.

* The figures show the amount of shortening in seconds.

No.	Name.	Age.	Duration of Lues	Eruption	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
15	J. W.	21	3 weeks	—	Normal		Normal	R. 12; L. 15
16	F. W.	18	5 days	+	Normal		Normal	Normal
17	M. L.	20	1 year	—	Radical Op. Right		R., Short'd; L. Norm.	R. 5; L. 8
18	T. S.	19	8 days	+	R. and L., O. M. S. C.	R. and L., Discharge	R. 10; L. 20	R. & L. length'd
19	F. H.	28	8 years	—	Normal	Normal	Normal	R. 15; L. 8
20	G. H.	28	2 months	—	Normal	Normal	Normal	R. 15; L. 12
21	L. K.	36	3 weeks	+	R., Drum destroyed; L., Cicatrices	R. and L., Discharge	R., Norm.; L. 50	R. 17; L. norm.
22	M. P.	25	4 weeks	+			Normal	R. 14; L. 8
23	J. H.	26	2 months	+			Normal	R. 17; L. 14
24	F. E.	20	8 days	—			Normal	R. 11; L. 8
25	R. R.	21	2 months	+			Normal	R. 13; L. 11
26	K. B.	36	12 years	+			Normal	R. 18; L. 15
27	H. P.	31	3 weeks	—			Normal	R. 5; L. 3
28	J. S.	42	3 weeks	+			Normal	Normal

29	B. R.	23	1 year	—?			Normal	R. 17; L. 9
30	H. S.	23	6 weeks	+			Normal	R. 13; L. 7
31	K. S.	24	3 weeks	—	R., Cicatrix; L., Adhesive processes	Previously	Normal	R. norm.; L. 13
32	K. R.	26	3 weeks	—			Normal	R. 5; L. 10
33	F. T.	30	4 weeks	—			Normal	Normal
34	V. B.	25	4 months	+	Scars R. and L.	Impaired hearing R. and L.	R. and L., Shortened	R. 10; L. 12
35.	R. S.	23	2 years	+	L., Chr. supp. healed.		R., Norm.; L., Short'd	R. 10; L. 5
36	J. M.	28	5 years	+			Normal	R. norm.; L. 5
37	A. W.	19	34 days	—			Normal	R. 10; L. 10
38	E. G.	23	5½ mos.	—	Normal		Normal	R. 17; L. 28
39	J. S.	31	3 weeks	—	Normal		Normal	Normal
40	A. H.	75	1 year	+	Normal		Normal	R. 5; L. 10
41	A. H.	28	3 years	+	Normal		Normal	Normal
42	J. M.	51	14 years	+	Normal	Vertigo and noises	Normal	R. 10; L. 15
43	A. A.	20	6 years	+	Normal		Normal	R. 10; L. 16

* The figures show the amount of shortening in seconds.

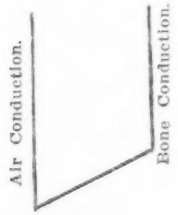
No.	Name.	Age.	Duration of Lues	Eruption	Otoscopic Findings	Aural Symptoms	Air Conduction	Bone Conduction*
44	J. R.	30	1 year	+	Retracted drums		Normal	Normal
45	L. L.	26	3 weeks	+	Normal		Normal	Normal
46	A. S.	32	3 weeks	—	Normal		Normal	Normal
47	J. S.	36	1 year	+	Normal		Normal	R. 5; L. 10
48	A. M.	27	1 month	—	Normal		Normal	R. 5; L. 5
49	H. H.	25	3 weeks	—	Normal		Normal	Normal
50	L. A.	20	2 weeks	—	Normal		Normal	Normal
51	M. Z.	32	6 weeks	+	Normal		Normal	R. 2; L. 6
52	R. F.	23	3 years	+	Normal		Normal	R. 15; L. 15
53	F. V.	19	8 weeks	+	Normal		Normal	R. 10; L. 10
54	J. S.	19	2 months	+	Normal		Normal	R. 15; L. 10
55	R. E.	15	6 weeks	—	Normal		Normal	R. 6; L. 6
56	M. J.	30	2 months	+	R. and L., Dry perf.	Impaired hearing	R. and L., Shortened	R. and L. norm.

* The figures show the amount of shortening in seconds.

iologic changes in the vessels are already present, leading to a shortened bone conduction.

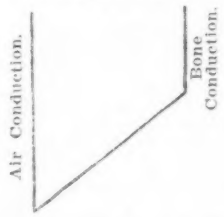
The following fact is evident: Although there are no exact and systematic tests which express mathematically the relationship between air and bone conduction in the normal and in the impaired ear, still we were convinced during our examination that bone conduction was decidedly reduced in luetic affection of the ear, and that a similar degree of shortening corresponded to a greater impairment of hearing in an ear whose internal ear was not affected by syphilis.

Figure 1-a.



Læsio auris internæ non-luetica.

Figure 1-b.



Læsio auris internæ luetica.

Naturally there are differences depending on the interpretation of each examiner, as mentioned above.

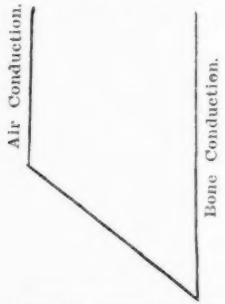
Of interest and of diagnostic value are the results of the tuning fork in patients with middle ear disease who have had syphilis. I shall briefly relate a case of this kind.

A young woman came to our clinic, complaining of various febrile symptoms, intense headache, and severe pulsating pain in the ear, which had lasted several days.

The drum membrane was diffusely reddened and swollen. No details were visible and the posterosuperior quadrant of the drum was bulging. Weber was lateralized to the affected ear, in which there was decided impairment of hearing. Air conduction was almost normal, while bone conduction in the bad ear, tested with a tuning fork of middle register, on the mastoid process, was surely not lengthened. I made a diagnosis of "acute middle ear suppuration, very probably in a syphilitic individual." The patient admitted the infection and the Wassermann was very positive. I was the more convinced of the presence of syphilis because the bone

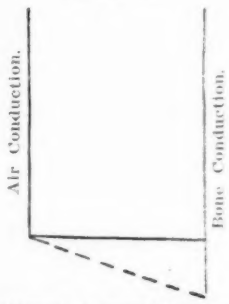
conduction was not lengthened, in spite of the fact that an acute otitis media was present. Therefore, there must have been a shortened bone conduction in the ear prior to the advent of the acute otitis; and the increased bone conduction consequential to the acute middle ear affection altered the picture from a shortened bone conduction to one of normal duration. To this assumption exception is naturally to be made in those cases where a shortened bone conduction is attributable to an affection of the internal ear, resulting from some injurious agent or due to a harmful vocation.

Figure 2-a.



Impediment to sound conduction in a non-luetic individual.

Figure 2-b.



Impediment to sound conduction in a luetic individual.

The question still remains to be answered as to the point of time after syphilitic infection at which this symptom, if it occurs at all, is elicited.

In very early cases presenting an initial lesion without local glandular involvement, and having a negative Wassermann, i. e., in patients in whom the diagnosis was based solely on the demonstration of spirochetæ in the primary sore, I have never observed a shortening. From these I have excluded those cases showing shortened bone conduction whose histories gave other etiologic factors. But in these cases shortened bone conduction is generally connected with an impairment of hearing. The symptom makes its appearance about the time when the local syphilis becomes general, approximately with the occurrence of the first papular rash; occasionally during the period when Wassermann becomes positive. The exact time cannot be fixed, for among my patients there are those who,

with normal hearing and without other demonstrable cause, showed a shortened bone conduction even before the serum reaction became positive; so that this symptom could be the first to awken the suspicion of a generalized syphilis. Patients were examined, also, who at the beginning of the eruption still had normal bone conduction and in whom a shortening was observed only after the rash had existed for quite some time.

On the other hand, a mechanic in whom a chancre was diagnosed after the demonstration of spirochetæ in the initial lesion, was sent to me for otologic examination preparatory to the injection of salvarsan. Wassermann negative; no glandular swelling or other sign of general syphilis. Since the bone conduction on both sides was shortened, although the patient had normal hearing, I believed, in opposition to the dermatologist, that I could made a diagnosis of already generalized syphilis.

The patient was treated with salvarsan and Hg., and for half a year there were neither clinical symptoms nor blood findings which pointed to syphilis. Bone conduction remained shortened. After six months papules appeared on the lips and tonsils, and Wassermann became positive. This case seems to indicate that shortened bone conduction in the presence of normal hearing may precede every other symptom of generalized lues.

I could never note any difference in the shortening before and after salvarsan or mercurial treatment. It is by no means easy to give a true explanation for this interesting condition. At the outset I thought I could find a connection between this symptom and a change in the blood pressure, but all experiments in this direction gave negative results. Consequently, it seemed necessary to determine whether shortened bone conduction was not to be found under other circumstances in individuals with unimpaired hearing. As is known, Wanner has claimed shortening of bone conduction to be characteristic of pathologic changes in the cranium or its contents. We know, also, that in tumors and in hydrocephalus, even without cochlear or vestibular symptoms, shortened bone conduction may be found. Such an observation was referred to by Ruttin in the Austrian Otological Society.

Herzog, in a recently published article in the *Münchener*

med. Wochenschrift, showed that in cases of growths of the dura to the bone, bone conduction could remain unaltered; indeed, it could be lengthened under certain circumstances. Besides in cases of tumor, hydrocephalus, and cerebral arteriosclerosis (Konrad Stein), I myself have been able to find this symptom unquestionably present in three cases of epilepsy and in one case of tetany. These observations, therefore, led to the conclusion that shortened bone conduction in the presence of normal hearing is indicative of a symptom which cannot be directly attributed to the ear itself, but is the result of the increased intracranial or intraspinal pressure.

In almost all of the recently published works dealing with the examination of the cerebrospinal fluid and the determination of its pressure in syphilis, a rise in the pressure was found by the respective authors. I have, therefore, in several cases measured the shortening of bone conduction before and after lumbar puncture. With the exception of one case, it was possible within three or four hours after lumbar puncture to establish the disappearance of the shortened bone conduction. I withdraw on the average five to six cubic centimeters. It was remarkable that in one case the shortening was greater two hours after the lumbar puncture than it had been prior thereto, and that only after another two hours did this shortening disappear. However, two or three days later the shortening was again to be found.

In spite of the fact that in this direction my investigations have not been very extensive, I believe that the shortening of bone conduction is to be attributed to the increase of the intracranial pressure, and that those cases of lues which do not present the symptom also show no increased cerebrospinal pressure.

If we admit this causative relationship between shortening of bone conduction and a rise in intracranial pressure, it also becomes clear why this symptom is as a rule first found when local syphilis becomes general. In the same manner may be explained the vertigo and disturbances of equilibrium during the second stage of florid lues, concerning which I made a communication at the Otological Congress at Frankfurt on Main. On this ground also may be understood the exaggeration of skin and tendon reflexes which Finger established years ago on the basis of fifty cases examined immediately

before and after the eruption of the rash. Remarkable, and especially worthy of mention, is the circumstance that occasionally we may determine a difference in the degree of shortening between the two sides of the same patient: that, for example, the shortened bone conduction of the right mastoid may be decidedly greater than that of the left mastoid.

I should like to describe briefly a particularly characteristic case:

T. M., twenty-five years old, kitchen maid. Three weeks ago initial lesion on the genitalia. Pale roseola on the trunk and extremities. Numerous large papules on the labia and at the anus. Lymphadenitis inguinalis. Wassermann positive.

Otoscopy.—Drum membrane and middle ear on both sides, normal. Bone conduction, measured with the electric tuning fork of Urbantschitsch, twice as short on the right side as on the left.

Nervous system.—On the left side distinct exaggeration of reflexes.

In this case, therefore, the greater shortening—that is, the greater intracranial pressure of the right side—is parallel with the exaggeration of the contralateral reflexes, a status which again points to the connection between intracranial pressure and shortening.

My chief, Prof. Urbantschitsch, has devised a tuning fork, which by the application of the electric current enables us to measure bone conduction exactly and to express it numerically. Prof. Urbantschitsch was kind enough to measure the bone conduction in a great number of luetics, and we were able here also in 80 per cent of the cases to confirm the above described findings. The exact construction of this instrument will be demonstrated by my chief on scientists' day in Vienna. In connection therewith I shall report the more exact findings of the difference in bone conduction between luetics and non-luetics, resulting from this method of examination.

LXX.

THE NASAL VOICE WITH REFERENCE TO ITS BEARING ON THE PRACTICE OF RHINO-LARYNGOLOGY.*

By Elmer L. Kenyon, A. B., M. D.,

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The special significance of the nasal voice to the rhino-laryngologist lies in this, that its correction is commonly dependent upon nasal or pharyngeal surgery, that under certain circumstances its presence modifies in an important manner the ordinary indications for operative treatment of the nose or nasopharynx, and that, without a knowledge of the nature and significance of the nasal voice, mistakes in diagnosis, prognosis and treatment are liable to be made, and operations of permanent injury to the patient undertaken.

The nature and cause of the nasal voice, or nasaling of the voice, is now, after many years of patient experimental study by men like Passavant, Merkel, Wintrich, Grützner, Schmidt and H. Gutzmann, quite satisfactorily established. One should rule out of mind nasal resonance as distinguished from nasaling, for a certain degree of nasal resonance is a quality of all normal voices, and is particularly significant in all voices of especial richness of quality. Nasaling, on the other hand, is not only due to an extreme exaggeration of the normal mechanical conditions upon which nasal resonance depends, but it is also produced, in the second of its two forms, by an entirely different mechanism from that responsible for nasal resonance, and consequently with a different voice effect.

The familiar nasaling which is produced by obstruction in the nose or in the nasopharynx affects primarily only the normally nasal sounds, altering their quality through impairment of the conditions of resonance in the nose and nasopharynx.

*Read before the Chicago Laryngological and Otological Society, May 27, 1913.

When in the production of the nasal sounds, m, n and ng, the vibrating air from the larynx is permitted by the low position of the soft palate to pass outward through an unobstructed nasopharyngeal cavity and through unobstructed nasal cavities, the resulting sounds are normally produced and sound normally nasal. When, however, the nasopharynx or nose is obstructed beyond a certain degree, these nasal sounds, while still nasal, take on an altered quality, sometimes spoken of as a "dead" quality. This alteration from the normal resonance of the nasal sounds produced by obstruction varies as to whether the obstruction be nasal or nasopharyngeal.

The kinds of obstruction in the nose and nasopharynx are known to every rhinologist and need not be considered here. One cause of rhinolalia clausa should, however, be mentioned. When we consider that rhinolalia clausa depends upon obstruction to the free passage of air from the pharynx through the nasopharynx and nose in the production of the nasal sounds, it is evident that such affections of the soft palate as bind the velum closely to the posterior wall must, by preventing the passage of air into the nasopharynx, also be responsible for rhinolalia clausa. This occurs, of course, chiefly from the adhesions following syphilitic diseases of the soft palate.

Rhinolalia clausa involves the speech not only with respect to the nasal sounds themselves, but also affects the sounds immediately preceding or following them, for during the normal production of many of these adjacent sounds the soft palate is still at least partially dropped, and the sounds are, therefore, to a certain degree, nasal. And, besides, the sounds normally oral may be irregularly involved, through their functional nasalization, in the manner to be described later (as rhinolalia aperta functionalis), owing to the exaggeration of the nasal qualities of the speech in general.

The second form of nasaling affects the mechanism for producing not the nasal but the oral sounds. Only one anatomic structure is at fault, the palate. If the palate is normally formed, and if the movements of the soft palate are normal, the velum leaps quickly backward and upward with each oral sound to meet the posterior wall of the pharynx, thus shutting off all or nearly all air from the nasopharynx and nose, and compelling it to pass out of the mouth. Any condition which produces either a permanent opening in the soft or

hard palate, or interferes with the movements of the soft palate, has the effect of preventing the relatively complete shutting off of air from the nasopharynx and nose when demanded in the formation of the oral sounds; the sound producing air then escapes freely into the nasopharynx and nose, and consequently transforms the oral into nasal sounds. If the opening from the mouth or pharynx through the hard or soft palate be in the posterior two-thirds of the space between mouth and posterior pharyngeal wall, all spoken sounds are nasal; if the opening occur, on the other hand, only in the anterior third of this distance, all sounds are apt to be normal excepting those normally produced at or near the lips, which then become nasal. In rhinolalia aperta then, when, as ordinarily is the case, dependent upon openings in the posterior two-thirds of the palate, all sounds are organically nasal, the usual nasal sounds m, n and ng being produced normally, and the oral sounds abnormally.

Rhinolalia aperta is dependent characteristically upon cleft palate. Likewise a congenitally short palate, being unable to reach the posterior wall on phonation, has identically the same effect on the voice. Destructive disease of the soft palate, often resulting from late syphilitic ulceration, is a quite common cause. And with the soft and hard palate normally formed, if the movements of the velum be interfered with, rhinolalia aperta follows as a necessary consequence. This occurs in deep infiltrations of the soft palate, chiefly syphilitic, in organic paralysis, notably bulbar paralysis, in paresis following diphtheria, and in paresis following rough handling of the palate in operations for adenoids.

A combination of the two organic causes of nasaling is not uncommon and complicates the situation. Rhinolalia mixta occurs when the nose or nasopharynx is occluded, and at the same time the palatal closure for the oral sounds is prevented in any of the ways mentioned. The voice partakes of the qualities of both forms of nasaling.

Thus far we have spoken only of organic causes of nasaling; but the functional causes for both forms are of particular importance to the rhinologist, and will not be neglected in a later portion of this article.

The diagnosis of rhinolalia clausa is not difficult for the rhinologist when it depends upon obvious extreme nasal or

nasopharyngeal obstruction. Difficulties arise, however, because the degree of obstruction capable of producing nasaling is hard always to estimate, and if one is not on his guard he may be operating unnecessarily to produce enlargement of the nasal or nasopharyngeal space, in order to correct a nasal voice in no way dependent upon occlusion, and of course without improvement of the voice quality following. Such operative procedures may indeed result in a decidedly increased degree of permanent voice impairment. Accurate diagnosis alone can prevent errors of this kind. To this end let the rhinologist determine, first of all, the presence or absence of rhinolalia aperta. This, if present, will direct his attention to the condition of the palate, which should ordinarily have remedial consideration, if impaired, before any existent nasal or nasopharyngeal obstruction.

Rhinolalia aperta may be distinguished quite certainly from rhinolalia clausa by the ear of the rhinologist, if it be sufficiently trained. But ordinarily this method of differential diagnosis is not certain enough. More than twenty years ago Herman Gutzmann devised the following simple but usually effective test for rhinolalia aperta. It is based upon the proven fact that, if the nose be tightly closed while the vowel sounds are being enunciated, the intensity of the nasaling is decidedly increased, whereas if the voice impairment be due to rhinolalia clausa, no change in the quality of the vowel sounds occurs from holding the nose. He has the patient repeat the vowel sounds "ah-e" several times, both with and without the closure of the end of the nose by compression with the thumb and finger, and notes the result in the sound. If there is no change in the quality of the sound the nasaling is not due to rhinolalia aperta, but if the nasaling is thereby increased the test proves the presence of rhinolalia aperta. The test is a delicate one. It is rendered more easily interpreted if the vowels be increased in number. A good series is ah-o-oo-e. This test may be supplemented by one which is perhaps still more delicate, but which demands some experience. The ear-tip at one end of a diagnostic ear tube is replaced by a nasal tip, and the tube is made to connect the naris of the patient with the ear of the physician. By this apparatus all sounds made with a lowered soft palate are greatly intensified, while the oral sounds are heard relatively much less loudly. With

the normal voice the nasal sounds and their immediate neighbors are alone intensified; in rhinolalia aperta all sounds, oral as well as nasal, are heard with about the same intensity. In this manner one may listen to spoken sounds directly as they emanate from the nose of the patient; to vowels (see the above series), to the speech of general conversation, or words especially provided for the test. For example, one may use a sentence which in the first half is made up of words each containing a nasal sound, and the last half containing only oral sounds, like the following: "In summer time, when men seem stricken under the strain of the sun, you or I or Frederick will probably be south of the Ohio." Through the nasal ear tube the latter half of the sentence is much less intense than the first half, if the voice be normal, and about equally intense in rhinolalia aperta.

If these tests show that rhinolalia aperta is absent, the nasaling must be due to nasal or nasopharyngeal obstruction. If they show that rhinolalia aperta is present, the diagnosis of any also existing rhinolalia clausa will depend upon the quality of the nasaling and upon the objective evidences of occlusion in the nose and nasopharynx. It is sometimes important, as will be seen later, to learn whether a rhinolalia clausa is dependent not upon obstruction in the nose or nasopharynx, but to an abnormal action of the soft palate. If the nose and nasopharynx are not totally occluded, this can be shown by the diagnostic nasal ear tube, for in such a case the nasal sounds will be greatly reduced in normal intensity, and probably no more intense than the oral sounds.

The application of a knowledge of nasaling in the practice of rhinolaryngology may perhaps be most clearly brought out by the study of certain concrete occurrences. As a preliminary practical step in the discussion, I may mention two recent cases of nasaling, one in a young man and the other in a young woman. The young man was operated for adenoids for no other reason than to improve his voice, but it was not improved by the operation. Upon examination the uppermost part of an adenoid mass was seen to be still present posterior to the choanæ. Rhinolalia aperta was absent. The patient being returned to his physician for reoperation, came back now with a normal voice. The nasaling in the case of the young woman is very marked; the adenoid operation was done

three years ago. Here, too, rhinolalia aperta is absent. The amount of adenoid tissue now present is large, and, besides, the nose is markedly occluded. The parents are much more troubled about the voice than about the obstructed breathing, but have not yet consented to return to their physician for further operative treatment, which, from the standpoint of the voice, must include the removal of obstruction in the nose as well as for that in the nasopharynx. The only inference from these cases is the need from the standpoint of the voice for a clean adenoid operation.

But nasopharyngeal operations may have with respect to the voice quite unexpected results. For example, the adenoids in a patient with a markedly nasal voice are completely and expertly removed, and yet the nasaling, both as to quality and intensity, remains unaltered. The nose is found to have but moderate obstruction, this being due to vasomotor swelling. In such a case the rhinologist, if not on his guard (the nasaling being the abnormality demanding correction), may, without further diagnostic study, proceed to operate on the nose. If he does so, this second operation will also be without result in correction of the nasal voice. More than twenty years ago Herman Gutzmann* related a case of exactly this kind occurring in a young German merchant who had gone the rounds of the European capitals seeking relief for an unpleasant nasal voice. Each rhinologist, apparently quite ignorant of more than one cause for nasaling, that of obstruction in the nose or nasopharynx, and concluding, therefore, that the nasopharynx being free, obstruction in the nose must be at fault, had found some new portion of the patient's utterly unoffending nasal organ to remove until the nasal space had become quite enormous. But not the slightest change had any of the numerous operations accomplished towards improvement of the voice. What had occurred was this: Earlier in life adenoids had so nearly taken the place of a closed soft palate that the velum had ceased to respond for the nasal sounds differently from its response for the oral sounds; thus it had remained during speech constantly raised against the posterior wall of the pharynx, not only for the oral but also for the nasal sounds, instead of dropping when a nasal sound came into the speech; and through the inculcation of habit this had gone on

*Von den verschiedenen Formen des Näselens, Berlin, 1891.

just the same after the adenoids had been removed. It was, then, a case of rhinolalia clausa functionalis. In Gutzmann's case two weeks of training served to entirely correct the nasaling for which the nose had been repeatedly and unnecessarily mutilated. Similar badly advised operations are not very uncommon, and the only way to prevent them is to be fortified with a knowledge of the etiology and diagnosis of the different forms of nasaling.

The other functional form of nasaling, rhinolalia aperta functionalis, is more common following adenoid operations than the form just cited. Owing to the practically complete obstruction of the air through the nose by reason of a large adenoid mass, the soft palate fails to raise for the oral sounds, but remains continuously in a dropped position. In certain cases the elevation is physically prevented by the low position of a part of the adenoid tissue. After the adenoids have been removed the palate still adheres to its habit of inaction, the oral sounds are spoken with a nasal quality, and diagnostic tests reveal the rhinolalia aperta. The incorrect action of the velum will continue and will tend to become more and more confirmed until the habit is corrected by training. A similar and well known effect, temporary, however, in duration, results when the palate has been roughly handled during the adenoid operation. The correction of functional rhinolalia of either form is much easier if carried out as soon as possible after discovery, rather than after the habit has become thoroughly confirmed by continuation for a long period of years. The establishment of a rule with rhinologists of examining their adenoid patients a few weeks after operation, having the speech especially in mind, would be a saving adjunct to this procedure.

If the palatal paresis following diphtheria continues through a number of weeks the same "habit paralysis" of the velum will be in danger of inculcation. For this reason Gutzmann advises faradization and massage of the palate, if the nasaling following diphtheria continues longer than two or three weeks. Other causes for rhinolalia aperta functionalis are found in the childhood imitation of the speech of cleft palate sufferers, and of other persons with a nasal voice, and also occasional cases of open nasaling due to habit, for which no explanation is discoverable.

The subject of the speech in cleft palate is exceedingly complex and exceedingly important, and is only to be touched upon in a paper of this character; but there are certain facts relating to the speech in every case of organic rhinolalia mixta which the rhinologist should always bear in mind. Since in cleft palate the nose is almost always obstructed, either by a deformed septum or enlarged turbinate bodies, or the nasopharynx is occluded by adenoids, cleft palate serves as a typical example of rhinolalia mixta. While the predominant nasaling of cleft palate is usually that of rhinolalia aperta due to imperfect closure of the palate, this is quite regularly combined with a quality of nasaling from rhinolalia clausa due to nasal or nasopharyngeal obstruction. Nature, indeed, shows a disposition to produce a vicarious palate by filling the nose with hypertrophied turbinate tissue. I remember years ago of seeing in an adult a wide palatal defect extending far forward into the hard palate, which cleft was literally completely filled with a massive lower turbinate body. The effect of this natural obturator was undoubtedly very favorable to the patient's speech. Not understanding this, however, at the time, I began to consider the most favorable means for removing the enormous turbinate body, an operation which as I know now would have done the patient much harm. Fortunately, he was wiser than I and did not return for further treatment. Formerly one could see more unoperated palatal clefts than he sees today, the rule being now to operate during the first months of life, and the practical problem of the rhinologist has to do chiefly with operated cleft palate. As to unoperated palatal cleft cases, let it be remembered that operative interference with conditions producing nasal or nasopharyngeal occlusion should be undertaken only after the closest consideration of the speech conditions involved.

But after operation for closure of the palatal cleft it usually happens either that the soft palate is too short to reach close to the posterior pharyngeal wall, or else the palate is incapable of sufficient movement to perform its natural function in speech. Consequently most cleft palate patients possess to a lesser or greater degree a permanent rhinolalia aperta voice, even after everything possible for the speech, both by operation and training, has been accomplished. In such cases nasal occlusion, as has been said, is with practical regularity present, and also adenoid tissue is common in the nasopharynx.

Moreover, in these cases there is much tendency to eustachian and middle ear inflammation, leading pretty regularly to more or less loss of hearing.

Since nasal occlusion and adenoids are associated in the mind of the rhinologist as a cause of aural inflammations, his disposition is naturally towards operative procedures upon the occluded nose and nasopharynx. It is at this point that considerations relating to the speech become important. In the first place, it is to be considered that other causes for aural inflammation exist in these cases beside those having reference to nasal or nasopharyngeal occlusion. This is proven by the fact that such aural inflammations go on after operative measures have been taken, and Gutzmann declares that they go on practically without any modification resulting from such operations. When one considers the permanent opening between the pharynx and nasopharynx with the continuous rasping effect of food and fluids, at any rate on the lower portion of the mucous membrane of the nasopharynx, one is not surprised that a tendency to middle ear inflammations exists even aside from that due to occlusive causes.

In rhinolalia mixta, whether from cleft palate or other causes, one should approach the question of operation for obstruction in the nose or nasopharynx with a great deal of care. If the rhinologist operates with the free hand usually employed, aiming to produce a normal breathing space, he will often be surprised to note the harm he has done to the voice. The work of training of months may be undone by a single operation of this kind. For in rhinolalia aperta a considerable amount of occlusion in the nose and nasopharynx improves the voice. The presence of adenoids is of the greatest advantage, especially during the early period of training. Thus one should always bear in mind that the presence of rhinolalia aperta serves in a measure to reverse the ordinary indications for nasal and nasopharyngeal operation for occlusion.

The following suggestions will serve to render the subject more concrete: First, the seeming demand for operation on the nose or nasopharynx, for the purpose of aiding in the correction of a tendency to inflammation of the ears, should be weighed very carefully against the demand of the speech for the continuance of nasopharyngeal and nasal occlusion; second, operation for the improvement of the breathing space through the nose should be extremely conservative, certainly

never producing a fully normal breathing space; third, operation for the elimination of the voice qualities of rhinolalia clausa should also be undertaken with the same extreme care, for a slightly obstructed voice is much more understandable than a badly open voice. And finally, operative effort on either nose or nasopharynx should proceed not by a single large operation, but step by step, with a waiting period between, so as to be able to call a quick halt if any signs of going too far become evident. Regarding the turbinates, extremely cautious cauterization would suit the conditions better than resection. Adenoids should be removed only after the clearest evidence of demand for removal of sufficient importance to outweigh the demands of the voice, and even then a partial operation should always be given preference over complete removal.

TREATMENT.

The correction of rhinolalia clausa functionalis involves the reinstitution of the habit of dropping of the soft palate for each nasal sound. The patient's ear should be educated to appreciate the quality of nasal as compared with oral sounds. He should be directed to take deep conscious nasal inhalations and exhalations, and then to make each nasal sound slowly and deeply through the nose. The appreciation of nasal quality tells him whether the air is or is not passing out of the nose; also the vibration of the bony nasal wall may be felt; but, better still, the nasal ear diagnostic tube serves this purpose. Having learned how to produce the individual nasal sounds, he then practices slowly and distinctly each vowel and certain regularly associated consonants with each nasal sound; and then individual words slowly spoken, and finally sentences.

The treatment of rhinolalia aperta functionalis aims to reestablish the raising of the soft palate against the posterior pharyngeal wall with each oral sound. Here, too, the patient should be trained to recognize the distinction between oral and nasal sounds. To this end deep breaths through the nose and through the mouth should be taken; nasal and oral sounds should be spoken freely before the patient in order to educate his ear. And the recognition of the difference between his own oral sounds when produced correctly and nasally may be aided by holding the palate back against the

posterior pharyngeal wall while oral sounds are produced. A moderately stiff flexible flat aluminum nasal applicator, so shaped as to follow the contour of the palate and with its tip wound with a soft pledget of cotton, answers the purpose admirably. The patient speaks certain selected sounds with the palate held back, and immediately repeats them without the aid of the palatal elevator, attempting to bring the sound out of the mouth rather than out of the nose.

This method serves also as a means for producing passive movement of the palate; and faradization may also be employed for this purpose. Both methods are impossible in persons with an especially sensitive mucous membrane. Active movement of the palate is brought about by the employment of selected vowel and combined vowel and consonant exercises. They should be spoken forcibly and explosively, and with an effort to direct the sound out of the mouth, and to obtain the desired oral quality. It is best at first to base these exercises upon the vowel sound Ah, which demands less perfect closure of the soft palate than the other vowels, using this sound in various consonant combinations with some success before proceeding to o, oo, aw, e and the others. Especial difficulties will require a variety of expedients. The prognosis is excellent, the length of treatment dependent upon the intelligence of the patient and the duration of the disorder.

To summarize:

1. The rhinologist is very liable to error in diagnosis, treatment and prognosis where nasaling is the disturbance in question, unless he is able to diagnose the manner of production and cause of the alteration of the voice in each particular case.

2. He should be on his guard for functional nasaling from either one of the two causes following operation for adenoids. Otherwise operations unnecessary and harmful may be done.

3. Operations on the nose or nasopharynx for nasal voice should not be undertaken until a clear understanding of the cause of the nasaling has been arrived at.

4. A defective palate, for example, when due to congenital palatal cleft, serves to in large measure reverse the ordinary indications for operation for nasal or nasopharyngeal occlusion, and unless the voice indications are carefully followed, serious and permanent injury to the speech may result from such operations.

LXXI.

CAISSON WORKERS' DEAFNESS.*

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Chicago.

This preliminary report of the form of occupation deafness occurring in workers under compressed air was undertaken at the suggestion of Dr. Peter Bassoe, who found in his investigations of caisson workers that a large percentage of them suffered from deafness of varying degrees.

I have examined thirteen workers under compressed air. The men who follow this class of work are mostly Irish in nationality. They are an easy-going sort of people, who make good wages and spend it as rapidly as it is earned. They are addicted to drink, as a rule. This no doubt increases the damage done to the cochlear nerve by their occupation.

The hearing tests were made oftentimes under unfavorable circumstances, being made at times in saloons, in boarding houses, and in other places where it was not possible to secure quiet for tests for the whispered voice. The whispered voice, under these circumstances, could hardly be heard more than four meters by a perfectly normal ear.

In the whisper test the numbers "24," "28," "22" were used for testing the lower pitches of the voice, and the numbers "66" and "67" for testing the higher pitches.

Bone conduction was tested with a large A Edelmann fork that vibrates normally three minutes when struck strongly.

Weber was also tested with this fork.

Rinné was tested with the c Hartmann fork of 256 vibrations. The other forks of the Hartmann set were also used. No reliance was placed on the C_1 and C_2 forks of this set, because of their richness in overtones. The low limit was determined by a set of C tuning forks giving 32, 64, 128, 256,

*Read before the Chicago Laryngological and Otological Society, May 27, 1913.

512, 1024 and 2048 d. v. per second. The lowest pitched fork heard is recorded as the low limit without endeavoring to determine more closely the exact limit.

A Galton-Edelmann whistle was used for testing the upper limit of hearing. The normal ear hears 25,000 vibrations, as registered by this whistle, or even a little higher.

When the caloric test was used it was by using irrigations of cold water.

The terms "blocked," "bends," "chokes," and "blind staggers," applied by the men to the various conditions under which they suffered, are probably to be interpreted as follows:

"Blocked" seems to mean a middle ear affection such as is seen in going up or down an elevator in a high office building, but of a greater degree. At times the term covers rupture of the drum membrane.

The term "bends" is applied to a painful condition of the extremities which Dr. Bassoe has found to be due to spinal cord and other lesions.

The term "chokes" is applied to a disturbance of respiration which the men refer to the neck.

"Blind staggers" appears to be a condition in which there is vertigo.

Case 1.—D. R., age fifty years. Has worked under compressed air for twenty-three years. The first time he went down he was "blocked" under thirty pounds pressure. He had nosebleed when he came out. He went under pressure very slowly because his ears were giving trouble.

Present Condition.—Right drum membrane considerably retracted, luster good, light reflex distorted, translucency about normal. Left drum membrane considerably retracted, light reflex preserved.

Functional Tests.—Whisper, right, "28" equals 1 m., "66" equals 50 m.; left, "24" equals 1.25 m., "67" equals 1 m. Bone conduction shortened fifteen seconds. Weber lateralized to the left ear. Rinné, right, slightly plus; left, slightly plus. Low limit, right, equals 64 d. v.; left, equals 64 d. v. High limit, right, equals 16,000 d. v.; left, equals 19,000 d. v. No spontaneous nystagmus.

Nose and Throat.—Right lower turbinate very large. Septum deflected to the left. Pharynx normal.

Diagnosis.—Chronic tubal tympanic catarrh. Labyrinthine deafness.

This case illustrates well what may happen to a caisson worker who has some condition predisposing to tubal occlusion. He had a deflected septum and enlarged lower turbinate, and the first time he entered the lock he was "blocked." The retracted drum membranes show that the tubal occlusion had lasted a considerable length of time.

The much shortened hearing distance for the low pitched whisper "28" and "24," and the elevation of the lower limit to 64 d. v., are evidence of a middle ear type of deafness which was no doubt intensified by the work under compressed air.

The shortened bone conduction, though common to advanced age, is doubtless a part of the labyrinthine deafness. The positive Rinné is marked evidence of labyrinthine deafness, for with a middle ear deafness of the amount present in this case there must needs be considerable labyrinthine deafness present to prevent the Rinné bcoming negative.

The lowering of the high limit of hearing from the normal of about 25,000 d. v. per second to 16,000 d. v. in the right ear, and 19,000 in the left ear, makes the diagnosis of labyrinthine deafness positive.

Case 2.—E. F., age fifty-three years; Irish; married. Twenty-four years ago patient worked under compressed air. A week after quitting work he felt a little nauseated in the evening. About 2 a. m. he became dizzy and, he thinks, totally deaf. After four months he recovered some hearing in the left ear. He is a bricklayer by occupation, but has been unable to follow this occupation since, on account of his impaired hearing. Following the attack of dizziness he stayed in bed three days. When he got up he walked like a half drunken man. He has had no other dizzy spells. He consulted a physician at the time, who said that he had a "rush of blood to the head." Some other physician told him that both ear drums were "cracked."

Present Condition.—Right drum membrane thickened, opaque, retracted, with an area of atrophy in front of the manubrium. Left drum membrane very strongly retracted, thickened and opaque.

Functional Tests.—Patient is apparently totally deaf in the right ear. He does not hear the voice, the tuning forks or the Edelmann whistle. Bone conduction, slightly prolonged. Weber lateralized to the left ear. Rinné, right, minus (heard in the

left ear); left, minus. Low limit, right, none; left, 2048. High limit, left, 14,000 d. v. Both vestibular systems respond normally to the caloric test.

Diagnosis.—Chronic tubal tympanic catarrh, right and left. Total labyrinthine deafness, right. Partial labyrinthine deafness, left.

This case illustrates what very serious labyrinthine disturbance may be brought about by compressed air. Thus, he developed a lesion of his vestibular apparatus a week after quitting work. He became totally deaf in the right ear and very largely deaf in the left ear. Whatever the lesion was that caused the nausea, dizziness and staggering gait, he appears to have completely recovered from it.

Case 3.—M. D., age fifty-six years; widower. Occupation, tunnel brick worker. He began to work under compressed air twenty-two years ago. He quit such work two years ago. He has often been "blocked." Denies venereal disease. Drinks very little.

Present Condition.—Right drum membrane about normal. Left drum membrane about normal.

Functional Tests.—Whisper, right, "24" and "66" equal 2 m.; left, "24" and "66" equal 2 m. Bone conduction shortened slightly. Weber not lateralized. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. Hears all the C forks. High limit, right, equals 19,000; left, equals 19,000. Nose and throat practically normal. Blood pressure 125 mm.

Diagnosis.—Labyrinthine deafness.

This patient has little or no middle ear deafness. The shortened hearing distance was due to the noisy surroundings under which the tests were made. On the other hand, bone conduction is shortened slightly, and the upper limit has fallen to 19,000 d. v. This degeneration in the cochlear nerve is not due to increased blood pressure, for the blood pressure is practically normal. This case illustrates typically the results on the labyrinth of working under compressed air.

Case 4.—M. G., age twenty-seven years. Patient has worked under compressed air for the last four or five years. He has had the "bends" three or four times. He has been "blocked" four or five times. Denies venereal disease. Drinks four or five drinks of whiskey a day and four or five bottles of beer.

Present Condition.—Right drum membrane normal. Left drum membrane normal in position, but luster deadened and color reddish, with a deeply injected streak along the manubrium. Septum of nose strongly deflected to the left, so that the left naris is completely occluded. Pharynx is dry and glazed.

Functional Tests.—Whisper, right, "24," "66" equal 60 cm.; left, "24," "66" equal close to the ear. Bone conduction apparently shortened. Weber lateralized to the left ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. All the other forks heard with each ear, but C, considerably shortened in the left ear. High limit, right, 24,000; left, 20,000.

Diagnosis.—Deflected septum; tubal occlusion. Labyrinthine deafness.

In this case the greater amount of middle ear deafness in the left ear, as shown by the diminished hearing distance ("24" equals close to the ear) and Weber lateralized in the left ear, is probably the result of an anatomic peculiarity (septum deviated to the left). It is not clear why there should be so much diminution in the hearing distance for the whispered voice, with a range of hearing in the right ear of from 32 d. v. to 24,000 d. v., and in the left ear of from 32 d. v. to 20,000 d. v. The comparatively short time he worked under compressed air probably accounts for the labyrinthine deafness being no greater.

Case 5.—A. C., age fifty-five years; married. Has been a worker under compressed air at intervals since the age of twenty-two. Complains of a buzzing noise in both ears. Was "blocked" several times twelve or fifteen years ago, and became dizzy. Denies venereal disease.

Present Condition.—Right drum membrane: Luster good, color good, position good. There is an area of atrophy in the anterior inferior quadrant. Left drum membrane normal in position and luster, but thickened and opaque. Septum deflected to the left. Left naris obstructed. Throat normal. Blood pressure 130 mm.

Functional Tests.—Whisper, right, "24" equals 3 m.; left, "24" equals 3½ m. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right equals 15,000 d. v.; left, equals 12,000 d. v. Bone conduction shortened about twenty seconds.

Weber lateralized to the right ear. Rinné, right, plus, but shortened; left, plus. Caloric test gives a weakened normal reaction.

Diagnosis.—Middle ear deafness on the right side. Bilateral labyrinthine deafness. Deflected septum.

This patient has quite good hearing for the low tones. His hearing for the low pitched whisper is practically normal. The marked shortening of bone conduction points toward a labyrinthine deafness, and this is made certain by the lowered upper limit, which is 15,000 d. v. on the right side and 12,000 d. v. on the left side. He seems to have had some disturbance of the vestibular nerve, as shown by the diminished caloric reaction. The dizziness he felt some twelve or fifteen years ago may have marked the time of the occurrence of the vestibular lesion. His blood pressure of 130 mm. is not abnormal for a man of fifty-five years.

Case 6.—P. H., age forty-eight years; widower. Denies venereal disease. Does not drink. At the age of twenty-five he began working under compressed air, and this occupation he followed for five years. He has often been "blocked" and had the "bends." He had an operation on the left side of his nose, with the idea of relieving trouble with his ear. This operation was not done until he had worked under compressed air nearly five years.

Present Condition.—Both external auditory canals were found filled with wax. After this was removed the right drum membrane was found to be about normal in luster and color. Transparency clear, but with a double light reflex. The left drum membrane was slightly dimmed in luster and moderately retracted. The left naris was occluded by a deflected septum. The pharynx was about normal.

Functional Tests.—Whisper, right, "24" and "66" equal 4 m.; left, "24" and "66" equal 1 m. Bone conduction shortened about twenty seconds. Weber lateralized in the right ear. Rinné, right, plus; left, plus (not heard at all by bone conduction). Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 20,000 d. v.; left, equals 16,000 d. v.

Diagnosis.—Deflected septum. Tubal occlusion. Labyrinthine deafness.

In this case the middle ear deafness on the left side was no doubt largely due to the nasal trouble on that side. This also

may be responsible for the greater degree of labyrinthine deafness on that side.

Case 7.—W. K., age forty-five years; Irish; single. Has worked under compressed air for twenty-two years. A part of the time he worked under a pressure of forty-eight pounds. He had "blind staggers" in 1911, lasting three hours. He had "blind staggers" also five years before that. He has had paraplegia from his work. He has been "blocked" many times, and has had the "bends" and the "chokes." He takes about five drinks of whiskey daily, and ten or twelve glasses of beer. He had gonorrhea three years ago, also soft chancre. Denies syphilis. He has had a buzzing tinnitus since a too rapid passage through the lock five years ago.

Present Condition.—Right drum membrane considerably retracted, luster dim. Left drum membrane moderately retracted, but luster fairly good. Nose and throat normal.

Functional Tests.—Whisper, right, not heard; left, hears "66" close to the ear. Conversational voice, right, "24" equals 60 cm.; left, "24" equals 1 m. Bone conduction shortened thirty seconds. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, Edelmann whistle not heard; left, equals 12,000 d. v. He hears all the C forks with both ears up to C_4, which he cannot hear with the right ear, though he hears it much shortened with the left ear. Caloric reaction normal.

Diagnosis.—Labyrinthine deafness.

The "blind staggers" may have been due to a disturbance in the vestibular system, but it has been completely recovered from. The continued use of alcohol no doubt contributed to the severity of the labyrinthine degeneration. Note the marked shortening of bone conduction (thirty seconds) and the lateralization of bone conduction in the left ear, which is the better hearing ear. Note, too, that though he hears 32 d. v. there is very marked lessening of air conduction for low sounds, inasmuch as he does not hear the whispered voice at all with the right ear. Note how much the upper limit of hearing has been lowered in the right ear (between C_3 and C_4).

Case 8.—M. L., age thirty-three years; Irish. Has worked under compressed air for five or six years, under a pressure of from five to twenty-five pounds. Has often been "blocked" in the left ear. Has often had an uncomfortable sensation in the

left frontal sinus. Has had a singing tinnitus in the left ear for three years.

Present Condition.—Right drum membrane about normal. Left drum membrane a little thickened, luster nearly normal. Nose, hypertrophic rhinitis on both sides.

Functional Tests.—Whisper, right, "24" and "66" equals 3 m.; left, "28" and "67" equals 15 cm. Bone conduction moderately shortened. Weber lateralized to the right (?). Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 25,000 left, equals 24,000.

Diagnosis.—Tubal tympanic catarrh.

This man has suffered but little from his work under compressed air. He seems to have had some predisposition to tubal occlusion on the left side.

Case 9.—T. D., age forty-six years. Now a saloon keeper. Worked under compressed air for two years at a pressure of twenty-four pounds. Was never "blocked."

Present Condition.—Right drum membrane normal. Left drum membrane strongly retracted. Had polypoid degeneration of right concha media. Has chronic rhinitis on both sides.

Functional Tests.—Bone conduction shortened ten seconds. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 26,000; left, equals 25,000.

Diagnosis.—Tubal tympanic catarrh.

This man's hearing was apparently but little affected by his working under compressed air. However, the time he worked was comparatively short.

Case 10.—J. M., age forty-two years; Irish; single. Worked under compressed air eighteen years ago for eight months at a pressure of twenty-two pounds. Was "blocked." He had bleeding from the nose and the left ear as he came out of the lock. He had severe pains shortly after he came out, and for twelve hours afterwards he was out of his head. He tried to jump out of the third story window the next night. This mental disturbance lasted for three or four days. He was dizzy, but could walk straight. He has a buzzing tinnitus in the left ear. He takes a glass of beer occasionally. Has had gonorrhea, but denies syphilis.

Present Condition.—Right drum membrane lusterless, but normal in position. Left drum membrane retracted, luster good. Throat normal. Septum deflected towards the right.

Functional Tests.—Whisper, right, "24" equals 30 cm., "66" equals 15 cm.; left, "24" and "66" equals loud and close to the ear. Bone conduction much shortened. Weber lateralized to the left ear. Low limit, right, hears 32 d. v.; left, hears 32 d. v. C_4 shortened in left ear. High limit, right, equals 20,000; left, equals 19,000. Irrigation of right ear with cold water gives rotary and horizontal nystagmus to the left. Irrigation of left ear with cold water gives rotary and horizontal nystagmus to the right, but chiefly rotary.

Diagnosis.—Tubal tympanic catarrh. Labyrinthine deafness.

The vestibular disturbance in this case must have been slight, for, although he was dizzy, he could walk straight. Since he suffered from mental disturbance, the lesion of the vestibular system may well have been central. In this case, as in others, there is a want of correlation between the hearing distance for low pitched sounds and the lower tone limit.

Case 11.—G. G., age sixty-nine years; Dane. Worked under compressed air for two or three years, usually under a pressure of twenty pounds. While working under thirty pounds pressure he had the "bends." He was "blocked" once. He has not worked under compresesd air for eighteen years.

Present Condition.—Right drum membrane slightly cloudy. Left drum membrane normal. Has rhinitis with a little crusting. His pharynx shows chronic pharyngitis.

Functional Tests.—Whisper, right, "28" and "66" equals 10 cm.; left, "24" equals 20 cm.; "67" equals 10 cm. Bone conduction shortened twenty seconds. Weber lateralized to the right ear. Rinné, right, plus; left, plus. Low limit, right, hears 64, but not 32 d. v.; left, hears 32 d. v. High limit, right, equals 19,000 d. v.; left, equals 19,000 d. v.

Diagnosis.—Chronic tubal tympanic catarrh. Labyrinthine deafness.

In this case the nasopharyngeal condition is probably the cause of the middle ear deafness present. The labyrinthine deafness may be the result of his advanced age.

Case 12.—I. H., age forty-six years; Norwegian. Worked under compressed air for two years at a pressure of from three to twenty-five pounds. He had the "bends" once while working under heavy pressure. This trouble came on three or four hours after getting out of the lock. The next day he could not work. He had to stay in bed for three days.

Present Condition.—Right drum membrane retracted, luster good, thin. Left drum membrane retracted, luster good, thin.

Functional Tests.—Whisper, right, "24" and "66" equals 4 m.; left, "24" equals 4 m.; "67" equals 3 m. Bone conduction normal. Weber lateralized to the left ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 25,000 d. v. left, equals 21,000 d. v.

Diagnosis.—Labyrinthine deafness.

This patient has not much change of a labyrinthine nature. A lowering of the high limit to 21,000 may accompany a middle ear deafness, but could hardly occur with a middle ear deafness of so slight a degree as to be manifested only by a lateralization of the Weber test.

Case 13.—P. J., age forty-five years; Irish. Worked under compressed air for seven years, at a pressure of from fourteen to thirty-two pounds. He was "blocked" several times.

Present Condition.—Right drum membrane not visible for wax. After removal of the wax it was seen to be slightly retracted, but of good luster. Left drum membrane retracted, luster good. Nose and throat about normal.

Functional Tests.—Whisper, right, "24" equals 60 cm.; "66" equals 60 cm.; left, "24" and "66" equals 3 m. Bone conduction slightly shortened. Weber lateralized to the right ear. Rinné, right, plus; left, plus. Low limit, right, hears 32 d. v.; left, hears 32 d. v. High limit, right, equals 15,000 d. v.; left, equals 16,000 d. v.

Diagnosis.—Labyrinthine deafness.

Here we have a marked labyrinthine deafness, as shown by the shortened bone conduction and lowering of the upper limit to 15,000 and 16,000 d. v., respectively. The patient's age is not sufficient to account for such a marked degeneration of the cochlear nerve as is shown by the tests.

Considering these cases as a whole, we may say that the results upon the ear of working under compressed air are comprised chiefly under three heads:

1. Tubal tympanic catarrh. This occurs principally in those who are predisposed to tubal occlusion by some peculiarity of nasal development; such as a deflected septum, hypertrophied turbinates, occlusion of the nares, etc. If a person with such malformation undertakes to work under compressed

air the symptoms due to his tubal occlusion are exaggerated and eventually become fixed.

2. Some workers under compressed air develop symptoms referable to the vestibular system, either in its central mechanism, in the course of the vestibular nerve, or in the terminal end organs. Whatever the lesion and wherever it is situated, the results are recovered from completely.

3. The most characteristic result of working under compressed air is a loss of a considerable portion of the upper range of hearing, with marked lessening of the duration of bone conduction. In a few cases the whole organ of Corti seems to have been destroyed, and that suddenly. This lesion occurred after coming out of the lock, in one case a week after. The cases of this sort can be explained on the supposition of a setting free of gases from the blood and their accumulation in the cochlear tube; or possibly a labyrinthitis was set up that destroyed the organ of Corti, or a neuritis, as Wittmack found in guinea pigs exposed to noise, poisons, etc. Whatever the explanation, the vestibular portion of the labyrinth is either more resistant to injury or else it has a greater power of recuperation. In none of the cases examined was there a failure to respond to the caloric test.

The most characteristic result of work under compressed air is a loss of the upper tone limit corresponding in type to the loss of hearing due to working in extremely noisy places—as, for instance, in boiler making shops. It appears to be a slow degeneration that, starting at the upper limit of hearing, gradually extends downwards. The amount of deafness of this type occurring in caisson workers corresponds to some extent to the time the patient has spent working under compressed air. The changes causing the loss of the upper limit of hearing do not seem to be limited to the basal coil of the cochlea, but extend throughout the whole cochlea, for there is marked shortening of bone conduction for the lower pitches, which are not lost.

The middle ear deafness that occurs in caisson workers may be prevented by removing the predisposing causes of tubal tympanic catarrh. The labyrinthine deafness that results from long continued work under compressed air is incurable. I see no way of preventing its development except by refraining from working under compressed air.

If deafness is to be minimized in these workers they should be examined before beginning work, to be sure that the nose is in a normal condition. They should abstain from alcohol. They should be examined from time to time to see whether labyrinthine degeneration has set in, and if it has, they should be urged to change occupation before the deafness has progressed far enough to interfere with other occupations.

The sudden lesions of the labyrinth are probably not preventable by any precautions that the men could be induced to take, beyond the slow decompression advised to prevent other lesions occurring in caisson workers.

LXXII.

THE MÉNIÈRE SYMPTOM-COMPLEX—A CLINICAL REVIEW.*

By Hill Hastings, M. D.,

Los Angeles.

I wish to present to you some case records of labyrinthine affections in which the Ménière symptom-complex occurred, to discuss those of special interest and to invite your discussion of the subject. I regret that I cannot hope to bring before you any new facts, nor do I presume to attempt a classification of nonsuppurative labyrinthine affections.

The subject of nonsuppurative affections of the labyrinth has lagged somewhat in comparison with that of labyrinthitis secondary to middle ear suppuration. There are two reasons why this should be true. First, suppuration of the labyrinth has offered a field wherein the clinical findings can often be verified by operation. Secondly, labyrinthine suppuration is not infrequently the cause of death. Autopsies are, therefore, frequently obtained. On the other hand, it is an exceedingly rare event to be able to verify by autopsy one's clinical observations in nonsuppurative affections of the labyrinth, death having resulted from other causes remotely related to the ear affection. For these reasons it is incumbent upon us as clinicians to review our past clinical experience, readjust our methods of examination in accordance with recent scientific investigations, and thus do what we can to illuminate by clinical experience the still dark portion of the ear. One must often regret that the hidden nerve field of the ear cannot be seen by the otologist as is the fundus of the eye by the ophthalmologist.

I have recently had occasion to review one hundred and forty-seven of my own records of nonsuppurative cases in

*Chairman's address, Western Section American Laryngological, Rhinological and Otological Society, Oakland, California, April 15, 1913.

which the functional tests showed involvement of the auditory nerve apparatus. Seventy-four cases (about 50 per cent) were without definite middle ear signs or symptoms. The remaining seventy-three cases showed signs of labyrinthine deafness, classified as follows: In otosclerosis, thirty-seven cases; in chronic catarrhal otitis media, twenty-three cases; in tuberculosis of the ear, thirteen cases. (Tubercular cases are included because the labyrinthine destruction did not follow the usual course found secondary to middle ear abscess.)

Of the seventy-four labyrinthine cases (not secondary to middle ear disease) twenty (27 per cent) presented Ménière's symptoms. In eight cases the dizzy spells were of the apoplectic form.

The term "Ménière's disease" has been rather loosely applied. The classical case, reported by Ménière in 1861, served to direct attention to the anatomic seat of a diseased condition that caused a characteristic combination of symptoms—deafness and dizziness, with nausea or vomiting. Ménière's interpretation of his findings in this case has been questioned. The hemorrhagic exudate, which he found in the labyrinth, could not in itself explain the cause of death. The patient, a girl of eighteen or nineteen years, died in five days, and the autopsy showed no middle ear suppuration and no suppurative intracranial lesion. Bezold believed that the case was one, not of hemorrhage in the labyrinth, but of epidemic cerebrospinal meningitis, with extension to the internal ear—a sporadic case in which the meningeal condition was overlooked. While this is the most plausible explanation of Ménière's case, the term "Ménière's disease" has been generally applied to nonsuppurative affections of the labyrinth or auditory nerve apparatus in which Ménière's symptom-complex occurs. And Ménière restricted the classification to cases in which nerve deafness and not middle ear deafness is found.

Cases presenting Ménière's symptom-complex as a rule result from (a) diseased conditions of the blood vessels supplying the labyrinth, or (b) disease of the eighth nerve, including its end organs in the labyrinth. From a therapeutic viewpoint, cases of Ménière's disease properly belong to the general practitioner or to the neurologist.

(A) Cases of the vascular class include: (1) Hemorrhagic or (2) serous exudates[16] in the labyrinth; (3) embolism of

the internal auditory artery or its branches (the terminal arteries are end arteries like those of the brain[1]); (4) embolism; (5) arteriosclerosis[15] (responsible for sudden anemia and possibly for nerve atrophy); (6) aneurismal pressure on the nerve; (7) leukemia (probably responsible for hemorrhagic exudates or nerve impoverishment; (8) severe anemias.

(B) Cases due primarily to neuritis include: (1) Toxic neuritis (salvarsan, alcohol, quinin, etc.); (2) infectious neuritis (syphilis, mumps, influenza, typhoid, tuberculosis, cerebrospinal meningitis, diabetes); (3) tumors of the eighth nerve itself and tumors pressing upon the eighth nerve (cerebellopontine angle—other nerves frequently affected at the same time); (4) meningitis with adhesions (other nerves frequently affected).

CASE REPORTS.

I. A Case of Ménière's Disease (Apoplectic Form) Occurring During Childbirth.—Mrs. W., age thirty-eight years, referred from San Diego on account of deafness in the right ear. The deafness was noticed after the birth of her last child, six months ago. Had never had any deafness or other ear trouble prior to that time. The labor was difficult. On coming out from the anesthetic she noticed great dizziness, nausea and vomiting. She did not attach any special significance to the symptoms; had never had similar experience in previous labors (has three healthy children). The dizziness continued two or three days, then subsided. Deafness was noticed on lying on her left ear. My examination showed complete nerve deafness. Weber to the good ear; bone conduction decreased; absolute deafness to whisper, speech, C to C_4 forks and to Galton whistle.

The etiologic relation to the labor, in this case, and the sudden onset, would indicate a hemorrhage or embolism of the cochlear branch of the artery. A toxic neuritis would be also a possible explanation. Lecompte[14] reports a similar case occurring in childbirth. H. Knapp[10] reports a similar case after miscarriage.

II. A Case of Ménière's Disease (Apoplectic Form); Atrophy of the Cochlear Nerve; No Permanent Injury to the Vestibular Nerve.—Miss S., age twenty-seven years, referred October 29, 1912, on account of deafness in right ear. Two

months ago, while making a purchase in a drug store, suddenly noticed a queer buzzing in the ear and sudden dizziness; had to "grab the counter to keep from falling"; did not lose consciousness; had to lie down; a doctor who was called said she had a "slight stroke." She was able to walk with help to a taxicab and went home; vomited in an hour, and off and on all day. After a day or two these symptoms passed off, but she noticed she was deaf. She did not have any headache or fever; the attack was not at the time of her menstrual flow, which had always been normal; her general health had been and is now excellent. Examination shows complete deafness in the right ear to acoumeter, speech, forks C (64 d. v.) to C_4, and to Galton; normal nystagmus reaction, caloric (hot and cold) and to turning.

I saw and reexamined this patient March 1, 1913, and found no changes from those above noted.

The suddenness of the attack, the complete deafness in the affected ear, without change in the vestibular reaction, and the absence of other signs or symptoms would indicate a hemorrhagic or serous exudate in the cochlea, resulting in complete atrophy of the organ of Corti; or blocking of the cochlear branch of the artery by an embolus.

III. A Case of Ménière's Disease (Apoplectic Form); Partial Deafness; No Vestibular Changes.—C. E. S., age thirty-six years, referred October 20, 1909, by Dr. Bryant, Glendale, on account of repeated attacks of dizziness and noises in the ear. His trouble dates back one year; began while walking on the street; suddenly fell backward and to the right; did not lose consciousness; was dizzy and nauseated for only a few minutes. He had a similar attack nine months ago, and another attack last night. Each attack is sudden, unheralded by any symptoms, and not accompanied by loss of consciousness. He always falls to the right. His family physician could find no diseased condition. He has never had syphilis; does not drink; general health is excellent.

Examination of ear, nose and throat is negative, except for bruised right auricle, due to last night's fall. The hearing in the left ear is slightly impaired; watch, one inch, ordinary conversation eight feet, bone conduction reduced twenty seconds (C 128 d. v.). Air conduction, c to c_4, reduced as follows: c, minus forty seconds, c_1, minus thirty seconds, c_2, minus

twenty seconds, c_3, minus thirty seconds, c_4, minus fifteen seconds. The patient says all the tones in the left ear are sharp as compared to the right. November 3, 1909, patient returns; has had another attack, similar in every way to the others. The dizziness lasted not over a minute, "had to grab the counter in the store to keep from falling—felt as if someone had bowled me over."

April, 1913, Dr. Bryant reports that the patient is still living and in excellent health; has moved up north, and has had only three attacks in the last two years. He states that the patient had a malarial history.

The cause in this case is, of course, obscure. The symptoms are suggestive of nerve irritation, from pressure periodically applied. The fact that the patient always fell to the right (the side of normal hearing) is contrary to my own experience in these cases. They have almost always fallen or had a tendency to fall to the diseased side.

IV. A Case of Mild Form of Ménière's Disease—Probably Tobacco Neuritis.—W. F. S., age forty-eight years, referred by Dr. Ross Reed, Pasadena, July 3, 1912, on account of sudden deafness and fullness in right ear and dizziness Trouble dates back one month; thought it was probably due to sudden blast from an automobile horn; has never had previous ear trouble; has not had a cold in the head; no catarrhal symptoms. General health good; denies syphilis; is married; has several children; does not drink, but has always smoked excessively, cigars and pipe; stopped smoking ten days ago.

The dizziness comes on at times; the spells are not severe enough to fall, but make him stagger; thinks the dizziness only comes when he moves his head; "the room seems to turn around."

Examination of the ear showed no middle ear signs. Hearing in right ear considerably reduced—acoumeter, ten inches; whisper ("four" and "six"), six inches and eighteen inches, respectively; conversation, twelve inches (dim); bone conduction reduced fifteen seconds; Gelle positive; air conduction, forks C to c_1 greatly reduced; c_2 and c_3 barely heard at all; c_1 poor; Galton whistle, questionable (hears with left ear); vestibular reaction to cold water and to turning is normal both sides. Examinaion of the eyes (Dr. L. W. Mansur) was negative. After five months' treatment this patient improved

somewhat. His last record (December 27, 1912) shows marked improvement in the hearing for forks, and moderate improvement to conversation—whisper, six feet; conversation, seven feet; forks, c. poor (barely heard); C. minus twenty-five seconds; c_2, minus twenty seconds; c_3, normal; c_4, normal. He had stopped smoking and had lived an easy life. He took about four ounces of potassium iodid, in five grain doses three times a day. I never could see the slightest improvement after catheterization of the tubes; in fact, inflation seemed to make him temporarily more deaf.

V. A Case of Nerve Deafness (Both Ears) Following an Attack of "Grippe"; Dizziness and Vomiting.—Mrs. W., age eighty-one years, referred October 23, 1912, on account of deafness. Two years ago patient had a severe attack of "grippe"—confined to her bed for five months; no meningeal symptoms; towards the end of the illness had severe vomiting spells with dizziness, attributed to the stomach; remembers that there were some noises in the ear; did not have earache nor ear discharge; became profoundly deaf. When she was able to get out of bed, noticed she could not walk in a straight line. Examination showed both drum membranes normal. Hearing to acoumeter, whisper and speech is gone; bone conduction (c 128 d. v.) reduced; forks C to c_4, negative in the right ear; in the left ear, could hear c_3 fork alone (thirty seconds); Galton not heard in either ear; vestibular reaction not tested.

J. M. Downey[17] reports a case of labyrinthine deafness due to "grippe," in which, in addition to the cochlear nerve atrophy, there was loss of vestibular reaction.

VI. Apoplectic Form of Ménière's Disease; Complete Deafness.—Mrs. A. S., age thirty-three years, referred April 21, 1910, by Dr. Pallette. Deafness in right ear dates back fifteen years. There was no earache and no discharge; remembers considerable dizziness and noises in the ear and that the hearing was suddenly lost and has never changed. For about ten years has had attacks of sudden dizziness, nausea and vomiting, coming on suddenly—so severe has fallen several times; last attack one and one-half years ago; the attack would not last long, and there was never any unconsciousness; attributed to the stomach.

Examination.—Middle ears normal. Left, hearing normal;

absolute deafness in the right ear to speech and all forks (C to c_4); Galton questionable (hears with good ear).

In this case the persistence of the attacks of dizziness, severe and sudden, over such a long period of time is noteworthy.

VII. A Case of Sudden Deafness, Dizziness and Vomiting.—C. F., age sixty-two years, referred by Dr. Bancroft, on account of deafness (left ear) of four days' duration. The deafness came on suddenly and without apparent cause. Noticed a "feeling of fullness in the ear and got very dizzy; had to lie down; did not fall; was nauseated and vomited." Did not have a cold; was not sick. Deafness in the right ear for years.

Examination.—Left ear, drum membrane thin, translucent, good mobility; no signs of middle ear inflammation. Hearing—acoumeter, one inch; whisper, twelve inches; speech, two feet; Rinné, positive; bone conduction reduced twenty-five seconds (C 128 fork); air conduction, c reduced fifteen seconds; c_1, fair; c_2, fair; c_3, fair; c_4, almost normal; Galton heard up to 25; vestibular reaction normal in both ears.

The age in this case would indicate a vascular change in the labyrinth, due likely to arteriosclerosis, and producing sudden anemia.

VIII. Sudden and Complete Nerve Deafness; Partial Restoration; Anemia. (?)—Mr. H. S., age fifty-seven years, referred April 30, 1910, from San Diego, by Dr. Fry. Complained of buzzing in both ears and deafness. Onset, four days ago; attributed to exposure to strong east wind and to removal of a heavy beard; thinks he caught cold, but has no signs of a recent cold in the nose, and no middle ear signs. Has always had good hearing in the right ear, but some deafness in the left ear for years. His hearing tests show complete absence of hearing in right ear to acoumeter, speech, and to forks C to c_4, and to Galton. In the left ear hearing greatly reduced—acoumeter, one-half inch; whisper, five inches; speech, three feet; forks, C, minus twenty seconds; c_1, good; c_2, good; c_3, good; c_4, not heard; Galton not heard; vestibular reaction, through turning, equal on each side, twenty seconds' duration.

May 4th (four days after my first examination) I was surprised to find that there was a return of hearing in the right

ear—acoumeter, negative; speech, six inches; C fork heard well; c_1, c_2 and c_3 fairly well heard, reduced about twenty seconds; c_4, not heard at all. The patient left for England. I referred him to an otologist in London. I never heard from this case. The complete deafness, sudden onset, and partial restoration of hearing, together with the age of the patient and hard radial arteries, indicated vascular disturbances in the labyrinthine vessels; possibly local anemia of the cochlea.

Richard Lake[15] reports several interesting cases of vertigo and deafness due to arteriosclerosis.

IX. A Case of Nerve Deafness; Apoplectic Form of Ménière's Disease; Ocular Paralysis.—E. P. M., age fifty-five years, a clinic patient, came to me first in November, 1904. Right ear deafness noticed three years before; came on suddenly; ear felt stuffed up and hissing noise in ear; no pain, no discharge; was working at his trade (blacksmith), but deafness was not apparently caused by any sudden noise or blow on the ear; no headaches; has been completely deaf in right ear since. Dizziness began six months ago, off and on; mild at first, but upon one occasion, fell down unconscious for a few seconds; got up and was ashamed for fear of being thought to be drunk (patient never drinks, not a glass of beer in two months on an average). In a week or two had another attack, preceded by a trembling; since then has had three or four attacks, but never quite lost consciousness. Last attack yesterday, preceded by increase in dizziness, but no other prodromic symptoms. Double vision, first noticed blurring and overlapping of images about seventeen years ago; gradually got worse, and for past five years has been severe; has to use a ground glass on left eye to enable to walk (consulted Dr. Pardee, San Francisco, who told him the nerve in the left eye was affected).

Previous History.—No illness since childhood; denies syphilis; general health is good; is married, has seven children.

Examination.—Middle ear condition is practically normal both sides. Hearing, complete deafness in right ear for all sounds.

In this case we have a record of ocular paralysis of seventeen years' duration, while the eighth nerve became affected only three years ago.

It has been my privilege recently to see at autopsy a brain from a case likely of this kind. The patient, a woman, age fifty years, had suffered for years with disturbances of the third, fifth and sixth nerves. The vessels at the base of the brain showed multiple aneurismal dilatations and marked atheromatous changes. Death had resulted from rupture of one of the aneurisms. The brain itself was normal. This patient had suffered from ocular paralysis for years. Diagnosis made only at autopsy.

X. A Case of Deafness and Dizziness, Due to "Nervous Prostration"—Deafness Almost Complete.—Mrs. B., age fifty-two years, came to my office October, 1912. Almost two years ago had a vomiting spell—deafness dates from that time. After this spell had "nervous prostration"—had been very nervous ever since; the last two months before coming to me patient had been getting better. After this spell there were many noises in the right ear—"severe noises." Following this attack was sick in bed six weeks; was dizzy and had a tendency to fall to the right. Had some slight confusion in memory. Had no difficulty in swallowing.

Examination.—Right ear drum dull and slightly retracted, but not sufficient to account for degree of deafness. Left ear—good glistening, large calcareous deposit below, but otherwise normal. Throat in good condition. Tube opened freely and drum membrane vibrated freely, if anything a little stiff. Hearing—right, absolutely destroyed; left, normal. It is noteworthy in these cases of unilateral nerve involvement, with vestibular irritation, that the tendency is to fall toward the affected side.

XI. A Case of Ménière's Disease (Apoplectic Form); Cochlear Nerve Atrophy.—Mrs. H., age fifty years, referred March 21, 1913, by Dr. A. L. Macleish, on account of deafness in left ear. Ear trouble dates back five weeks; began with dizziness, nausea and vomiting; attributed to the stomach; was so dizzy she had to go to bed; whenever she raised up would fall forward and to the left. Dizziness and nausea lasted three days. No earache, no discharge, no fever. One year ago, after "grippe," had some stuffiness in the same ear, relieved by two or three inflations. Is positive her hearing was acute up to this sudden attack, five weeks ago. Patient has been out of bed the past two weeks; is weak and uncer-

tain in her gait, but the dizziness has gone. Examination of the eyes (by Dr. A. L. Macleish) shows glaucoma, but no ocular paralysis. Blood examination (by Dr. Walter Brem) is negative; Wassermann reaction is negative. Aural examination—Left drum membrane practically normal; completely deaf to acoumeter, whisper, conversation, and to all forks, C to c_4, and to Galton. Caloric reaction (left) quickly developed (cold water, 66 degrees, one-half quart in left ear as against one quart in right ear); reaction nystagmus well marked, lasted thirty seconds, and accompanied by considerable nausea.

It was impossible to explain the etiology in this case. Syphilis and leukemia were excluded. The patient left for her home in the East, referred to her former consultant, Dr. Charles H. Beard, of Chicago.

Spaulding[16] has recently reported a similar case from supposed labyrinthine effusion, similar to iritis serosa or episcleritis.

XII. A Case of Nerve Deafness Associated With "Nervous Prostration"; Dizziness.—Mr. M. E. J., age fifty-two years, referred January 20, 1913, by Dr. Robert Lewis, Jr., New York. Has come to California for his health. No organic disease has been found, but has had a general nervous breakdown from long period of work as railroad man. Denies syphilis. Wassermann reaction (taken at request of Dr. Lewis) was negative. Deafness has been marked of late; rapidly grown worse in the right ear; almost complete deafness in the left ear. Has frequent dizzy spells, attributed to constipation; thinks he always tends to fall backward during the spells; "room seems to be turning around." Never any headache.

Examination.—Left membrane somewhat retracted, otherwise normal; right membrane slightly retracted. Hearing, right ear: acoumeter, one inch; whisper, three feet; speech, two feet (dim); Rinné, positive; forks C to c_4, reduced twenty-five to forty seconds; Galton heard up to 45. Hearing, left ear: absolute deafness to whisper, conversation and all forks (C to c_1) except c_3, which is faintly heard; Galton heard up to 40. Vestibular reaction is normal and equal both sides.

XIII. A Case of Atrophy of Both Cochlear Nerves; History of Hemorrhages and Dizziness.—Mrs. F. R., age fifty-

two years, referred from Pomona, December 19, 1910, on account of deafness both ears. Right ear had been deaf for five years. Left ear suddenly became deaf one month ago. At present unable to hear conversation in either ear. Right side deafness began five years ago with a severe attack of dizziness and general exhaustion. Was confined to her bed for six weeks. Vomited blood and had hemorrhages from the bowels off and on for two or three months, considerable prostration. (Unable to secure any opinion as to the nature of the disease.) From this attack right ear was rendered absolutely deaf, but could hear well with left ear until one month ago. Has had no earache or ear discharge, and nothing to account for the sudden deafness. Dizziness during the first attack was so severe had to sit down to keep from falling.

Examination.—Right drum membrane dull, slight retraction, otherwise negative. Left, the same. Hearing entirely gone in right ear, to watch, acoumeter, whisper, speech, and all forks; bone concduction (c 128) reduced to twenty seconds. Hearing left ear also entirely destroyed, except for the c 128 fork, which is heard about twenty seconds. Galton not heard in either ear. Pupils react normally to light and accommodation. No paralysis of the fourth, fifth, sixth or seventh nerve. Ears had been inflated for a month without any improvement in the hearing. Tube was open and dry, and no middle ear signs to account for the deafness. Patient was married and had several children; was not questioned as to specific history.

The peculiar attack, in which there was hemorrhage from the bowels and vomiting of blood for two or three months, might have a bearing on the etiology of this case. Patient was treated in another part of the country. If a careful diagnosis had been made at the time of the hemorrhage the cause for the nerve atrophy might have been found.

ETIOLOGY.

As to neuritis of the auditory nerve, some interesting facts are established. Neuritis probably occurs more frequently than one would think, and is more easily overlooked. Wittmaack[1] states that "the cochlear branch with its associated ganglion is imbedded in an unyielding bony capsule, partly surrounded by broad lymphatic spaces"; that "the blood ves-

sels are end arteries; that the ganglion cells are smaller than all other ganglion cells in the vertebrates; that, moreover, they are bipolar and possess medullary membranes; that these anatomic relations show the elective vulnerability of the cochlear nerve."

Paul Manasse[2] in thirty-one cases of eighth nerve deafness found that the cochlear branch alone was affected in twenty-four cases. Wittmaack states: "It is not strange that a particular group of nerve fibers should be affected. This can be observed in other nerves, i. e., the recurrent laryngeal nerve, also the optic nerve—as in tobacco amblyopia; also in neuritis from lead poisoning." So with eighth nerve disease the cochlear branch alone is often affected.

Wittmaack states: "It is presumable that in the labyrinth pathologic processes generally occur in the form of small morbid foci, in the form of small hemorrhages, or inflammatory exudates, or infiltrates, situated within the delicate membranous labyrinth. From many of the autopsy reports of cases diagnosed "Ménière's" it seems to me that a serous exudate or a sudden disturbance in the lymphatic circulation may have produced the symptoms." (Case XI may be explained in this way.) "Arteriosclerosis may produce nerve atrophy through disturbance in the nutrition of the nerve. Atrophy and neuritis cannot be differentiated clinically."

One fact stands out prominently, namely, that while optic neuritis and atrophy can be diagnosed objectively, auditory neuritis and atrophy can only be diagnosed during the life of the patient by testing the cochlear and vestibular nerves. That the clinical findings in such cases point to underlying conditions of grave concern to the patient should stimulate us to examine our ear cases most carefully.

Reports in the last year or two of neuritis occurring in syphilitics after salvarsan injections, show the auditory nerve affected about twice as often as the optic nerve. As to whether the syphilitic virus or the salvarsan is responsible for the neuritis cases, it is worth mentioning that Prof. Alexander, in six years prior to the advent of salvarsan, observed sixty-eight cases of lues of the ear; in only twelve cases symptoms appeared in the early stage (third to sixth month); whereas Prof. Finger in six months had as many cases after salvarsan treatment. Alexander, therefore, concluded that there must

be an etiologic relation in eighth nerve neuritis to salvarsan injections.

A large number of reports are being made of neurorecurrence after salvarsan injections. For example, Duel (Boston meeting, International Otological Congress, August, 1912) stated that he had seen three cases lately of eighth nerve neuritis after salvarsan injections—all within the first two months—an occurrence that he had not met with in an experience of fifteen years. He mentions that Benario collected fourteen thousand cases treated by salvarsan, in which one hundred and twenty-six cases of neurorecurrence occurred. Of the one hundred and twenty-six cases, fifty-one showed eighth nerve involvement.

THE THERAPY OF MÉNIÈRE'S DISEASE.

The therapeutic measures advocated in cases of Ménière's disease have been many and of remarkable variety.

Parry[5 9] advocated the use of a seton placed in a fold of tissues of the neck at the base of the skull.

Blake and Putnam[5 6 7], following Babinsky, used lumbar puncture with improvement in some cases. Dundas Grant,[4] Bradley,[11] and others, following Charcot, advised use of quinin in Ménière's disease. The use of pilocarpin, potassium iodid, complete body rest, depletion by sweating, and cathartics, is rational and advised by many authorities. The use of vaccines and bacterins, as reported by one observer (Sherman[8]), seems irrational, and his results not conclusive.

Where the labyrinth tests show complete nerve deafness of long duration, atrophy of the nerve has taken place, and treatment would, of course, be of no value in clearing up the condition; however, as the other cochlear nerve occasionally becomes affected, it is wise to attempt to ward off trouble by using those therapeutic measures applicable to the particular case. In acute cases during the course of syphilis specific treatment is, of course, to be instituted. As to whether or not salvarsan should be used is too large a question to be discussed in this paper. It seems to me that the answer to the question largely depends upon whether the threatened damage from the specific infection is great enough to warrant running the possible risk of the toxic damage from salvarsan.

In an acute case of Ménière's disease, not specific in origin,

a search should be made for the underlying cause, such as arteriosclerosis or endocarditis (from which a vegetation might have been broken off into the blood stream and lodged in the labyrinthine artery); or for some of the diseased blood conditions, as pernicious anemia, leukemia, etc.; or diabetes, nephritis, etc. As the treatment depends largely on treating the underlying condition, the otologist in most cases likely would best serve the patient by referring him to a good internist or neurologist, being content with pointing out the nerve change that his examination disclosed.

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LXXIII.

MALIGNANT TUMORS OF THE NASOPHARYNX, WITH REPORT OF CASES.*

By A. L. Kelsey, M. D., and J. M. Brown, M. D.,

Los Angeles.

An exhaustive and scientific study of malignant tumors of the nasopharynx from the literature available presents many difficulties and is indeed probably impossible.

In the first place the nasopharynx is quite inaccessible for purposes of diagnosis and operative procedures; then the widespread belief that the removal of a portion of a tumor will hasten a fatal issue has undoubtedly deterred many from availing themselves of the help which can be had from the microscope; and finally the classification of malignant tumors presents some moot questions, and there is yet a lack of unanimity among pathologists as to what class a given tumor should be placed in.

There seems to be a strong movement to classify endothelioma distinctly by itself, but still it is, at times, called a form of sarcoma, while at others it is placed under the carcinomata. Again, certain malignant tumors present mixed histologic elements, while inflammatory tissue in connection with these tumors may prove a pitfall in diagnosis.

Certain benign growths may also undergo malignant changes, as is shown by many authoritative reports, and should be properly classed as some particular form of malignancy.

If, as is often undoubtedly the case, a sufficiently large mass is not removed for examination, the pathologist is unable to make a correct diagnosis, because the sections available do not show the malignant elements, which may exist in other

*Read before a joint meeting of the Western Section of the American Oto-Rhinological and Laryngological Society and the Oto-Laryngological Section of the California Medical Society, April 5, 1913.

parts of the tumor. (This happened in one of our cases at the first examination.)

Early literature shows a mass of reports of nasopharyngeal growths, mostly loosely classed as polyps of some form, and is valueless to a study of the subject because of the lack of pathologic reports.

Undoubtedly a considerable number of cases have not been reported as yet. We recall several occurring in Los Angeles which are not used in this paper, but which will be reported later.

Chevalier Jackson in a paper published in 1901, dealing with carcinoma of the nasopharynx, quotes Moritz Schmidt as saying that in 32,997 nose and throat cases he had not seen a single case of primary carcinoma of the nasopharynx, and that Bosworth had not been able to gather more than six cases up to 1889, one being his own.

It is significant that Jackson, in the twelve years succeeding Bosworth's report, was able to add eight more cases, including one of his own, and that we, in a rather limited experience, are able to present one case, occurring in the last six months.

It would seem that many cases of carcinoma and other malignant tumors had been reported as having their origin in other regions, when they really may have sprung from the nasopharynx, and that many cases had been reported under misleading titles. In any case, later reports seem to show an increasing number of cases, and also a tendency to a more concise classification.

We do not feel ourselves competent at this time to approach the subject with the intention of writing a complete and scientific thesis, but will be content with presenting some facts gleaned from literature referred to by the Surgeon General's Reports, the Index Medicus, the Index of Otolaryngology, and such other papers as we have been able to find. We will present our own cases, four in number, which really gives us the one adequate excuse for this paper.

For the purposes of this study we submit the following classification of malignant tumors which may occur in the nasopharynx:

Carcinomata;—Glandular and squamous.

Sarcomata: Morphologically—Round celled; spindle celled; giant celled; mixed celled.

By origin—Fibrosarcoma; osteosarcoma; chondrosarcoma; myosarcoma.

Endotheliomata:—Lymphangio-endothelioma; hemangio-endothelioma.

From all available reported cases we deduce that these tumors occur with about the following relative frequency: Carcinoma, 30 per cent; sarcoma, 60 per cent; endothelioma, 10 per cent.

We are inclined to believe that these percentages will later be altered so that the endotheliomata will appear in greater numbrs, because the later reports undoubtedly show a much higher frequency than formerly, due to a more accurate classification which places some of the sarcomata and carcinomata properly under the head of endotheliomata.

Age.—Many authoritative reports indicate that malignant tumors occur in the various organs at a much earlier age than was formerly held.

Defour cites four cases of carcinoma, including one of the larynx, under one year of age.

Reliu, one of larynx under three years.

McKenzie, two cases under three years.

Rehn, one of larynx under three years.

Brem, one of cheek under one year.

Moritz Schmidt reports one case of the nasopharynx at fourteen, and Fauvel one case under seven. This we believe to be the youngest age in which carcinoma of this region has been noted.

We call attention to these reports of the early appearance of malignant tumors in other regions with the idea of inciting a more careful investigation of growths of the nasopharynx in the young, which might result in placing some of the so-called benign growths under a malignant classification.

Dabney, in 1912, cited a case of fibroma in a boy of fourteen years, which suddenly became sarcomatous. Sections of this tumor were taken at random, which showed it to be a true fibroma, but after its removal it recurred as a sarcoma. He claims that these tumors, having their period of activity during the period of development, and undergoing atrophy thereafter, or becoming malignant, show a probable common origin for these tumors and malignant growths. However, reports

available for this discussion show that the various tumors occur at about the following ages:

Carcinoma ..... 60 per cent between 40 and 60
Sarcoma..... 60 per cent between 10 and 30
Endothelioma.... 75 per cent between 20 and 40

Sex.—The male sex seem to largely predominate, especially so with endothelioma, which shows 75 per cent to occur in males.

Heredity.—All collected cases show a very small percentage giving a history of the occurrence of malignancy in the family. Of the fourteen cases of carcinoma reported by Chevalier Jackson, only one had a positive family history.

Location.—It is often difficult or impossible to determine with accuracy the actual origin of growths of the nasopharynx during life, especially when they are of considerable size, which is often the case when first seen, so that reports must often be looked upon with some doubt. Of cases gathered, when any mention is made of the origin, it would seem that they mostly spring from the vault or base of the sphenoid, while a smaller percentage occur in the lateral wall. A few cases are reported as being placed on the posterior end of the septum.

Symptoms.—Nose.—Nasal obstruction, due primarily to interference with circulation, producing turgescence of the mucous membrane, and later to the fact that the tumor closes the postnasal space. Discharge increases with the nasal turgescence, and is at first a thin mucus with no particular odor, but when the tumor becomes infected, it becomes purulent and characteristically fetid. At times it may be blood tinged. Hemorrhage is not very frequent in carcinoma or endothelioma, but fairly common in sarcoma.

Ears.—Deafness, with or without tinnitus, is present to some extent in nearly every case, and is of the eustachian tube variety. These symptoms are peculiarly apt to appear in a severe form when the tumor is situated on the lateral wall, and particularly when it has infiltrated the adjacent tissues, instead of projecting into the nasopharyngeal space, as is usually the case. One of our cases, however, showed no deafness, owing to the fact, no doubt, that the mass, though large, was rounded in form, well outlined and showed no signs of infiltration of the walls of the nasopharynx.

Pain.—Pain is present in the majority of cases early or late, and is located in the corresponding side of the head or throat. At times a certain branch of the fifth nerve is involved alone, while at other times more than one branch is irritated. In some cases the gasserian ganglion has borne the brunt of the invasion, and upon its removal has been found to be malignant. In a few cases such findings have prompted the diagnosis of primary malignancy of this ganglion, and the real origin of the tumor, which was in the nasopharynx, overlooked.

Glands.—Glandular enlargement always occurs early in carcinoma, but the enlargement may not be noticeable until rather late. In sarcoma it is not constant, and is a late development. In endothelioma it is early, and the glands become of great size. The glandular involvement is not by any means always due to malignant invasion, but quite often is a simple adenitis. It involves both deep and superficial glands.

Fixation of the lower jaw is apt to occur as a late symptom, when the tumor infiltrates the lateral wall rather than projecting into the nasopharynx, as is usually the case, and is due to involvement of the pterygoid muscle.

Retraction of the soft palate when at rest is a symptom mentioned by Trotter, due to infiltration of the levator palati muscle, and was observed in one of our cases.

Diagnosis.—Any patient showing one or more of the nasal symptoms enumerated, and particularly when coupled with enlargement of the glands, should have the nasopharynx examined at once. If a tumor or thickening is found, suspicion should be aroused, and when possible a portion of the tumor removed for a thorough examination by a pathologist. In no other way can a final and correct decision be had, and the patient is certainly entitled to have this decision given him as early in the course of the disease as possible.

Prognosis.—The prognosis is invariably bad, though two or three cases have been reported of nonrecurrence after several months, and one after three years.

Treatment.—Nonoperative.—One case is reported where the tumor disappeared after using radium, but the patient died six months later of meningitis, leading us to believe that the tumor, though apparently gone from the nasopharynx, was really progressing and had finally invaded the brain.

The injection of alcohol, formalin, adrenalin, and Coly's fluid has been tried with little success, only two cases being reported cured; one from the use of alcohol, and one by the use of formalin. Ligation of carotid artery is credited with one cured case.

It would seem that any medical treatment might be used logically in the late cases, with the expectation of reducing the size of the mass, and thus giving some relief to the patient, but not with any hope of cure.

Operative.—Many palliative and radical operations have been done by various methods, but the final results have proven very inadequate. Of the radical methods the ones most in use are the resection of the superior or inferior maxilla, splitting of the soft palate, with or without resection of the hard palate, and resecting the external nose, so that it may be pushed to one side.

Rhinologists, generally speaking, have stood against radical operations and favor conservative procedures, such as curettage, because it has been found that a large percentage of patients radically operated have not survived the operation very long.

The following cases were seen by us in our clinic in the Los Angeles Medical Department of the University of California:

Case 1. (May 6, 1907.)—Chinaman, age sixty years. He had been complaining for some months of a rather free watery discharge, without odor, and occlusion of the nose, especially the left side. Examination showed a rather soft mass in the nasopharynx, which bled slightly on touch. It seemed to be attached to the left lateral wall, and could be seen projecting somewhat into the nasal space. There was turgescence of the nasal mucous membrane of both sides. The cervical glands were slightly enlarged, with no special tenderness.

The nature of the case was not suspected at the time of the examination, and as much of the mass as could be reached was removed by the snare and curette through the nose, without much bleeding. His symptoms were greatly improved, but seen some weeks later it was found that the growth had returned, and his condition was even worse than before. A specimen was now submitted to Dr. S. T. Black, university

pathologist, who reported the tumor to be a sarcoma. Died at County Hospital, November 7, 1907.

Case 2. (January 8, 1913.)—Mrs. M., age about forty-eight years. History given by patient is as follows: A subject of tonsillitis all her life, very severe during early years. Three years ago a physician punctured a small cyst situated behind the right tonsil, which contained a serous-like fluid. Two or three weeks later the tonsil became swollen and highly inflamed, and she was confined to her bed for two or three weeks from what was thought to be an infection of the punctured cyst. A year and a half ago the cervical glands of the right side began to enlarge, without tenderness or pain, but ceased to enlarge six months ago. Two months ago she felt as if she had a cold, but there was no discharge. Since that there has been continuous obstruction of the nose. At no time has she had any pain, deafness or nasal discharge.

Examination shows the following conditions: Eyes, normal. Nose, patulous; only slight increase of mucus. Both choanæ seen to be obstructed by a large mass. Tonsils both larger than normal. Many cervical glands enlarged on both sides; one on right side the size of a small hen's egg, but no tenderness present.

The finger in the nasopharynx feels a firm mass entirely filling that space, slightly movable and apparently attached by a rather broad base to the posterior wall.

Corresponding to the right choana the tumor presents an area which feels velvety, and at this site is felt a small bud-like projection which was easily detached. Near this was a membrane which also came away, giving much better breathing space.

The removed portions were submitted to Drs. W. V. Brem and A. H. Zeiler, the university pathologists, who reported it as carcinoma.

The patient is cheerful and content, and refuses any operative interference. She was seen only once since the examination, and presented no new symptoms.

Case 3. (November 26, 1912.)—N. P., age thirty-seven; male; Greek. Family history negative. Personal history: Patient says he had syphilis fifteen years ago, and was treated for it several times since then, but has never had any symptoms of the disease.

History.—For six months he has been troubled with an excessive secretion from both nares and the nasopharynx, and for the same period he has had a gradually increasing nasal obstruction. The last month he has been compelled to breathe through his mouth only, and has lost twenty pounds in weight. Deafness has been present for about three months, at first intermittent, and lately constant.

Examination.—Ears, both tympanic membranes decidedly retracted and lusterless. Nose, lower and middle turbinates turgescent, and the nasal space full of mucus. No odor. With adrenalin the parts shrink to normal size, and a grayish mass is to be seen on both sides in the nasopharynx. On palpation a mass that nearly fills the nasopharynx can be made out. The base seems attached to the under surface of the sphenoid in the position of the pharyngeal tonsil, and spreads out latterly and downward in the walls of the pharynx. The glands of the neck in the anterior triangle on both sides were enormously enlarged.

A specimen of the tumor was secured and sent to the university pathologists, Drs. W. V. Brem and A. H. Zeiler, who reported a hemangio-endothelioma. A Wassermann test showed negative results.

On October 17, 1912, under ether, the mass was thoroughly curetted away, the operation giving rise to no great amount of hemorrhage.

The patient was seen March 26, 1913, when there was considerable mucopurulent discharge having some odor. He had been working hard during the months succeeding the operation and was feeling well. Palpation showed no return of the tumor. The cervical glands were somewhat larger.

On April 7, 1913, he was seen again, when it was found that there was a return of the growth, and he showed marked cachexia and decided increase in size of the cervical glands.

Case 4. (January 28, 1911.)—B. C., age thirty-five years; male; Austrian. Family history negative. Personal history negative. No syphilis.

History.—For six months he has noticed pain in and around the right ear. At first it was intermittent or neuralgic in character and very severe at times. Latterly it has been almost constant. For four months he has noticed a gradually increasing tinnitus, but deafness was not particularly noticed.

Nasal obstruction has been present for about one month, and limited to the right side.

Examination.—Tympanic membrane on the right side is decidedly retracted and lusterless. Left side normal. Nose negative. A mass high up on the right side of the nasopharynx can be seen with the mirror. On palpation a fairly hard mass the size of a pigeon's egg, and attached to the sphenoid, was felt. A small piece was removed and sent to the university laboratory for examination. The pathologist reported sarcoma. This patient disappeared from the clinic at this stage, so the subsequent history is not known.

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LXXIV.

INFLAMMATION OF THE LATERAL COLUMNS OF THE PHARYNX LEADING TO ABSCESS FORMATION, WITH REPORT OF CASES.*

By Henry L. Swain, M. D.,

New Haven.

When a tissue like the lymphoid structures in the nasopharynx and throat, possessed of certain peculiar characteristics, becomes inflamed in any one of its various portions, it might reasonably be expected that certain results would follow. Or, to put it in another way, if certain kinds of inflammation were possible in any one part of Waldeyer's ring, it certainly ought logically to be possible in any other portion. Any isolated mass of lymphoid tissue could, therefore, in a general way, be expected to act, when inflamed, exactly after the fashion of the faucial tonsil, and could have, like the latter, simple acute, follicular, rheumatic, diphtheritic and phlegmonous inflammations. As knowledge became more widespread concerning what happened to the adenoid or pharyngeal tonsil, we learned to know, as years went by, that it might have acute as well as chronic inflammation. The same is true of the lingual tonsil and at a meeting of our association, in 1898, at Pittsburg, it was my pleasure and privilege to read a paper chronicling various cases of acute inflammation of the lingual tonsil which ended in abscess formation.

In recent years much has been written in the journals concerning inflammation of the lateral columns of the pharynx, and I have a presentiment that if we continue to remove root and branch from our young children all of their adenoids and faucial tonsils, there will be abundant need for us to de-

*Read at the thirty-fifth meeting of the American Laryngological Association, held in Washington, May 5th to 7th, 1913, as a part of a discussion on Phlegmons of the Upper Air Passages.

vote increasingly more and more attention to these masses of lymphoid tissue as well as to the lingual tonsil. Hundreds of observations exist to attest to the fact that when, in years gone by, adenoids were removed in young children and the faucial tonsils were left untouched, the latter subsequently enlarged into a perfectly healthy growth, as though needed by the system, and as though trying to supply something to satisfy this lack or want. If they, the faucials, are nowadays also to be ruthlessly removed, there will, just as surely as the sun rises and sets, be enlargement of the lingual tonsil or lateral columns of the pharynx chronicled, as there has been of the faucials following the older simple adenoid operation. And if this is logical and true, we should find not only acute inflammation of these structures, but also the phlegmonous.

What was true way back in 1896, before the war upon the adenoid tissue was as general and as destructive as in recent years, is of course, as you all can bear me out, perfectly true of the present time. Not only have you and I been seeing cases of quinsy of the lingual tonsil from time to time, but it has been my fortune to see several within the last few months. In one of the last cases there was a great similarity noted, which was suggested in my earlier paper, to Ludwig's angina. There was here, curiously enough, enormous swelling of the submaxillary glands, inability to shut the mouth, owing to the swelling in the floor of the mouth and elevation and protrusion of the tongue. It was impossible to get the mouth open far enough to admit a finger, still less to make an examination with the mirror, and through no skill of mine the diagnosis was finally made, after the worst danger was over, when the swelling subsided, owing to rupture of the abscess into the throat. Examination of the lingual tonsil, when possible, revealed the spot of rupture to one side of the center. What was of even more importance, in a way, as illustrating the danger which the patient ran, was the observation of a subsiding swelling of the epiglottis and aryepiglottic fold which went way around to and included the arytenoids. The patient had, therefore, been in extremely threatening danger by reason of the edema of the glottis and could have readily died from suffocation, as has been frequently observed to be the case in real Ludwig's angina.

This latter possibility actually occurred only a week pre-

viously in the care of one of my ablest colleagues, who was operating to open the abscess of a genuine Ludwig's angina. He was compelled to make a brilliant emergency tracheotomy, rescuing the patient from immediate death. When respiration was restored he proceeded to find the abscess and drained it splendidly, only to lose the patient some days afterwards by sudden hemorrhage, of unknown origin, into the windpipe.

These two cases were immediately followed, in my own experience, by the last case that I have observed of abscess of the lateral column of the pharynx, the kind of phlegmon which I wish particularly to emphasize in this communication.

We have assumed more or less arbitrarily that as the lateral column of the pharynx is no mean mass of lymphoid tissue, and as it has been known to have all of the other kinds of inflammation to which lymphoid tissue is heir, we have a logical right to expect that there might also be the phlegmonous type, and I propose to submit the facts concerning certain cases I have observed to prove that such may be the case. If this is, however, as logical as I have indicated, it would seem strange that no one would have thought it worth while mentioning, unless, as I shrewdly suspect, few have believed it really to occur. Even so patient and persistent a writer and observer as Levinstein, of Berlin, who has with great detail told about all kinds of inflammation in the anginas of the lateral columns, as he calls them, fails to say anything definite about such a quinsy. I have not found it mentioned anywhere. Indeed, few of the textbooks think it is even necessary to mention the lateral column of the pharynx, probably reasoning that it would behave as any other lymphoid tissue did, and what was the use of talking about it? Moure, of Bordeaux, in his book comes as near to what I am considering as anybody whom I have happened to discover. He is very thorough in his pictures and in his descriptions of the anatomic relations of the faucial tonsil, and says that he finds a great many times the abscesses of the faucial tonsil point posteriorly.

Take this body of gentlemen present and listening to these words of mine, representing the best thought and broadest erudition of any group of men in the country in this particular line of work which we have before us, and if abscess of the lateral columns of the pharynx was of frequent occurrence

it would seem as though you must have all seen it many times. I am sure you will all, if you think a moment, recall cases of quinsy where you had tried to puncture and failed, which burst in the night, and nothing showing in front, you concluded it must have perforated posteriorly. Indeed, in some instances you have been able to absolutely observe the pus coming down from behind the palate. I myself have had numerous occasions where I have been unable to get at the abscess and have contented myself with the idea that the pus had not yet formed, only to later be forced to acknowledge that it was there all the time, and that in following the routine plan of making incisions I did not get to the spot where the pus was. It is easy enough if you have a pocket of pus and it shows white under the epithelial coating to know enough to cut in there and evacuate it. And that was just how I stumbled upon my first case of abscess of the lateral column of the pharynx. And then others followed, and I now have six or seven "sure enough" cases, my last one being within a week, as I have said, in a lad of thirteen.

As the subjective and local symptoms of all of these cases were about alike, it will not be necessary now to detain you but a very few minutes to describe what happened in one case which is doubly interesting in the fact that, like quinsies of the faucial tonsils, this of the lateral column seemed to have a tendency to recur. All of my cases have been in young people. All of them have had edema (except this last case of the boy of thirteen), and the edema has, in the case of the recurrent quinsy of the lateral column, been of the most threatening type.

In the case of a young student in our university, whose history I am about to relate, there seems to be no doubt that we have had three genuine occurrences of abscess in the same situation, and I am responsible for the fact that two of them were in the lateral column of the pharynx, in the same region, and on the same side.

The patient, a young man of nineteen, had been in my charge, at our infirmary, for two weeks with an active double otitis media. One side, the left, had subsided quickly; the other, being much more active, gave us much more trouble to control. At the end of the second week he suddenly had a very sharp rise of temperature and a chill. He broke out with

the most profuse erythematous eruption that I have ever seen. The body was completely covered with an urticaria, as it proved to be, and the first thought was that we had to do with a very active case of scarlet fever. With the beginning of the hives he had a very marked case of indigestion, if one chooses to use the term. The patient was nauseated, vomited and could retain nothing on his stomach for forty-eight hours. In the middle of this he began to have a sore throat. This latter symptom was, owing to the various other things which had to be done for him, somewhat neglected. It had been going on in a mild degree for two days before it assumed so active proportions that we must needs pay attention to it. It was on the left side, the side on which the ear, fortunately, seemed to be entirely through wth its acute stage. The tonsil of that side swelled and began to bulge out into the throat, being forced forward and downward toward the median line in quite the typical way for quinsy, if one did not observe carefully the situation. Being somewhat hoarse on the third day of his apparent quinsy, we discovered that he had a very marked edema of the aryepiglottic fold of that side. The arytenoid of that side was swollen as large as your thumb, a very large, baggy swelling. At the time of the discovery of the edema we twice attempted to find where the abscess was, before the fact was revealed that the tonsil was not swollen in the way which it usually is, or at least not displaced in the way in which it usually is, and that there was considerable space between the palate and the posterior pharyngeal wall. Poking forward the posterior pillar, I discovered that the lateral column of the pharynx was enormously swollen, and with a great deal of effort (for, as is usual, the mouth opened with great difficulty and one could see in only through a very small aperture) we hooked the palate forward sufficiently to discover that the upper part of the column of the pharynx was very much swollen and pushed toward the palate. Taking my cue from two previous cases of abscess in this region which I had formerly had, I made the incision around up in back of the palate into the swollen area and evacuated a large amount of pus. At this time it was learned from his parents, who had come upon the scene, that he had on a previous occasion been troubled with a sore throat where he had the utmost difficulty one night in breathing, and they

feared that he would suffocate, and where relief had come by the rupture of the abscess. No observations had been made as to where the abscess formed, but from my description of the situation as it was then, the mother felt very sure that the conditions had been exactly the same in the previous attack. In any case, they had not been able to see the place where the pus came from, following its evacuation. With the subsidence of the swelling and the retreat of the edema the urticaria vanished and the patient rapidly got well. During these days of observation which immediately followed the abscess I was able to absolutely ascertain that the opening was in the substance of the lateral column.

A year and a half later I was called to the infirmary to see this same boy, reporting that he had another attack of quinsy, which had not gone on so long untreated as before, in which there was just the appearance again of edematous swelling about the arytenoid of that side. But knowing from the previous history just where to go to get at the pus pocket, within twelve hours of the onset of the severe symptoms we located and opened the abscess, and as nearly as I can tell you it was in precisely the same spot.

It is interesting to chronicle that the hives were also present in a most profuse way, and that the patient suffered with an upset digestive tract.

Commenting on this case many interesting points occur to us. That the skin could have, together with the other tissues, what amounted almost to an edema, would seem to indicate that possibly the edema of the larynx in this case was also constitutional or systemic in origin. And this leads to the statement that the whole matter of edema of the larynx would seem to need a fresh rehearsal. It would be interesting to have us all, in the next few years, observe carefully our cases of edema and to see if we could establish some theory of their existence which is less open to objection than the old venous stasis theory which has generally been held when one had a foot, or a hand, or a leg where one desired to make clear why the swelling took place.

The investigations of Fisher, of Cleveland, have led him to believe that the edema is, in a sense, a local acidosis, and the cells of the small blood vessel walls allow of the escape of fluid into the tissues which in a state of health they do not

permit. No theory of pressure or damming back of the circulation could explain, to my mind at least, why a region like the aryepiglottic fold, which receives blood from an entirely different set of vessels from those which supply the tonsil and whose lymphatics evacuate very much lower into the neck, could, by any pressure or stasis which the abscess in the tonsil itself could make, be made edematous when so far away and so out of all immediate contact with the active purulent process. Hence one could in this case quite readily accept Fisher's idea that there has been brought about in some way this local acidosis, although it is still difficult to understand why the local acidosis should be found in the aryepiglottic fold and not in some other portion of the body, assuming as we do that there is no immediate direct connection in the circulation, venous or lymphatic, between the two areas.

Now I have recently, in this season, had in my own experience six cases of edema of the larynx. In two of them there was no quinsy at all. There was inflammation of the lateral column of the pharynx in both these instances, and there was in one a most marked general—what may be called rheumatic—infection, where various joints of the body were swollen and inflamed, with rapid jumps from these to other joints, and with no persistence of the symptoms in any one place for any length of time. In three other cases, in other years, mumps seemed to be the cause of the edema of the laryngeal region, which removes the cause of pressure or stasis or infection still farther away from the larynx than in any case of quinsy or inflammation of the faucial tonsils or lateral column. So that from these various cases of edema one has to look to some other cause than simple pressure, and the general acidosis of the system which had taken place in this case of lateral column phlegmon on these two occasions seems to afford a reasonable explanation of why he could have edema in any place in the body. If the system had been so disturbed as to produce this rise of temperature, erythematous urticaria, and upset digestion, it could also make the swelling in the region of the larynx. We have still to explain why it occurred first upon the side of the inflammation in the lateral column and secondly on the other side to a slighter degree.

Examination of the urine in this case showed high acidity, large indican reaction, and no albumin or sugar.

The well known curious vagaries of the socalled angioneurotic edema point all too clearly to the fact that local areas of edema can take place in almost any part of the body, even where there is no systemic or kidney condition to be looked upon as the causal agent.

The second interesting thing which we might dwell upon for a moment in this case is the fact of the recurrence on the same side, in essentially the same region, just exactly as we have so frequently observed in quinsies of the faucial tonsil.

The third point is one which anyone desiring to argue could well maintain—was this not, after all, an abscess of the tonsil which simply pointed posteriorly, and in the process of so doing caused infiltration and swelling of the lateral column of the pharynx? The only answer that I have to make to this is that I did see here the swelling and abscess so intimately in the substance of the lateral column that I do not believe it was a quinsy of the faucial tonsil. My reason for asserting that the more flatly is the fact that in these six cases of socalled abscess of the lateral column, the swelling or the displacement of the tonsil has been really different from that usually observed in true quinsy cases. To my mind, in the true quinsy case there is almost always swelling of the tonsil itself as well as displacement of it forwards and downwards toward the median line. In these marked cases of abscess of the lateral column the pillars of the palate, the tonsil, and the whole soft palate are all displaced, but there is almost no swelling of the tonsil itself, and the posterior pillar of the palate is pushed forward and away from the pharynx wall; whereas, when abscess occurs in the dead space above and laterally from the faucial tonsil, the usual peritonsillar abscess, there is almost always a pressure backward of the posterior pillar of the palate, bringing it into more immediate contact with the posterior pharyngeal wall than would be the case with the opposite side of the throat. So that if I have rightly observed the situation, I am almost sure that there is a pronounced difference in the swelling in the two instances which one can readily observe if he carefully watches the position of the posterior pillar of the palate.

Of the other cases that I have to chronicle, one was in a young lady of twenty, who had never had a quinsy to her knowledge before, which was the first case that I had observed

in whom there was also edema of the glottis, and where I made three distinct attempts to discover the abscess by puncturing into the peritonsillar space in the usual situation, only to discover that I had not been observing the case properly and that the abscess formation was really back of the palate. Here it was absolutely forced upon me by seeing the abscess practically protruding around the edge of the palate when the patient gagged, and the evacuation of the abscess was followed by the usual quick relief.

Moure speaks also of abscesses occurring in the substance of the posterior pillar of the palate. This I have also seen, and it is to my mind and eye a very different situation from what I am trying to describe as existing in the substance of the lateral column.

Between the two attacks in the patient first mentioned, I saw another student with the mumps who developed a supposed quinsy, but the real abscess formation was in the lateral column of the pharynx, as in these other instances, and he had a very profound edema which was most threatening in its aspects.

The fifth case was in a young man of twenty-two, with nothing particular to distinguish it from the others, the symptoms being all the same as in the case of the young lady first observed. It was a second abscess, the previous one having been in the usual situation back of the tonsil on the opposite side.

The last case, which I have so recently seen, was in the lad of thirteen, who had never had a previous known attack of quinsy, and where the lateral column was enormously swollen at the start. His tonsils had been removed, were very small, and only a little swollen during this attack. The first puncture of the lateral column brought out no pus, and I thought I had made a mistake in my diagnosis, and on the second occasion tried to find pus in the usual region, but failed also here. In the night the abscess ruptured, and the point of evacuation was in the lateral column, out of sight of direct vision, behind the palate.

There is nothing so particularly urgent in this presentment that should necessarily claim your attention for so long a time in a meeting of this kind, only the general interest which we all take in peculiar and rare phenomena, and the hope that

I have that in having mentioned this form of abscess, not only will some case be clearer to those of you who are here, but that perhaps the profession in general may by observing a little closer make their earlier incisions more effective, and this, as sometimes in my own cases I know, may save the patient from twelve to twenty-four hours of suffering, not to mention the relief to the pressure which in some way is causing an edema of the larynx. And a word here: whereas all these cases of edema have transpired without any actual serious interference with breathing or the symptoms being what might actually be called dangerous to life, yet I cannot contemplate the existence of a large edematous swelling on one or both sides of the larynx without feeling that the patient is seriously in danger of suffocation, and as every other thing one hears of cases that have ended fatally because of profound swelling, the possibility of locating the abscess a little earlier is one which may perhaps be a life saving device. In any case, I am grateful to you for the attention that has been accorded the paper.

SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of April 22, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Paper: Case of Compound Follicular Odontoma of Superior Maxilla.*

BY L. W. DEAN, M. D.

DISCUSSION.

DR. M. HERZOG said he knew comparatively little about such tumors. In general terms he might say that odontoma, as a rule, originates from embryonal inclusion. According to the paper, these tumors are quite rarely reported in man. However, reference was also made to those tumors occurring in lower animals. In the horse they are comparatively common compared with the human race, and he had examined probably four to six cases in horses. The speaker was sorry that no sections had been demonstrated by the essayist, because that would have been to him the most interesting part—to see how they were composed, and whether they contained epithelial cysts and gland-like structures. He could not say very much regarding these tumors in the human.

DR. JOSEPH C. BECK said there was one fact that ought to be brought out, namely, the use of the radiogram in the diagnosis. He recently had a case of dentigerous cyst with a retained tooth that was malformed. The condition had been treated for four years for an antrum suppuration, with an opening at the last molar region. Of course, the case was treated from a different point of view by the dentist. The X-ray picture showed very clearly a double partition of the socalled antrum, and a tooth in this dentigerous cyst or cavity. Another inter-

*See page 1077.

esting fact in this case was that during the operation, which was performed under general anesthesia, while prying the tooth loose it slipped. Three days later the tooth was removed from the nasal cavity. It had been forced through the two thin partitions into the antrum first and on into the nasal cavity.

Dr. Norval H. Pierce said that the division of these cases into inflammatory and embryologic cysts was very practical from a clinical viewpoint. All of the subdivisions given by the doctor could be embraced under these two heads—the embryologic and the inflammatory. The inflammatory is the socalled dentigerous cyst that we most frequently see. It is very frequently seen as a very small protuberance or cyst at the root of a tooth, when it has been removed for some cause or other, the cyst not having been suspected before. This cyst is capable of growing to an enormous extent, filling up the entire maxillary antrum.

The differential diagnosis between these cysts and empyema of the maxillary sinus is that the cysts contain cholesterin crystals.

Dr. Pierce had seen just one case of the embryologic form of cyst, and that was hardly a cyst. It occurred in a man who was brought to the Illinois Eye and Ear Infirmary, and he had a cleft reaching from just above the ala of the nose to the tear duct. In fact, the patient was sent from the eye clinic to the ear clinic, the man having gone to the eye department on account of difficulty with the tear apparatus. There was a bridge of normal bone separating the cleft above from that below, and the speaker could very easily detect two teeth just below the eye, and, running the probe down into the fistula below, which ran down underneath the gum, there was a cavity which apparently was filled with embryologic teeth. Of course, it is readily understood that by feeling with the probe one can differentiate between bone and teeth. The man only came once. He was told to come back for operation, but never appeared again. No X-ray picture was made. This is the only case that Dr. Pierce had seen of the embryologic form of dentigerous cyst.

Dr. Dean, in closing, said that no X-ray examination was made in this case because the diagnosis was plain. The sinus in the alveolus, from which the rudimentary teeth were being discharged, made a positive diagnosis. Dental surgeons have

not definitely decided as to whether it is possible to have a complete absence of a tooth of the second dentition, or its embryonic structure. In New York recently they had a case in which there was apparently a congenital absence of a tooth. The patient was seen by Dr. Ottolengui, who made a radiographic examination. The X-ray picture showed most beautifully a composite odontomata. This differs from the case which he had just cited in that the rudimentary teeth were solidly cemented together by cementum. This patient was suffering no inconvenience whatever.

Atresia of the External Canal.

DR. ALFRED LEWY showed a case of atresia of the external canal. The ear began discharging at the age of eighteen and ran for several years, but was finally cured. The woman was a patient of Dr. McBean, and was first seen by him last August. At that time there was almost complete atresia. After that she was not seen until a week or two ago, when she came in with complete atresia. The patient also has Raynaud's disease and scleroderma. (Since the reporting of the case the atresia has been cured apparently by electrolysis and the insertion of Bernays' splint.

Paper: Case of Meningitis in Which the Cisterna Magna Was Drained.*

BY NORVAL H. PIERCE, M. D.

Failures and Successes in Diagnosis and Surgical Intervention of Some Intracranial Diseases, Especially From the Standpoint of an Otolaryngologist, With Report of Cases.

(ABSTRACT.)

DR. JOSEPH C. BECK'S report included the following conditions: (1) Sinus thrombosis; (2) meningitis; (3) extradural abscess; (4) brain abscess; (5) brain tumor; (6) hypophysis tumor; (7) intracranial hemorrhage, with and without fracture of the skull; (8) Gasserian ganglion (intractable tic douloureaux); (9) external hydrocephalus; (10) encephalocele. Of each variety he cited at least one case, discussing such points as were new and helpful in making correct diag-

*See page 1081.

nosis, and dwelling upon the latest form of treatment. He also gave a summary of all cases treated, with a brief resume of points helpful in brain surgery. Discussion of symptoms, pathology and other well-known matters pertaining to this subject were omitted.

Of thirty-eight cases of sinus thrombosis, either with or without complications, as bulb and jugular involvement, meningitis, brain abscess and general sepsis, or some other general condition, as pneumonia in nephritis, etc., twenty-six recovered following operation. Of the twelve fatal cases, ten were complicated mostly by septic pneumonia, which came practically moribund to the operating table; the two remaining cases, which were diagnosed early and apparently not complicated, died from rapidly developing meningitis. Both of these cases were of the streptococcic type of infection. His experience with meningitis was limited to seven cases, which he operated on by the method suggested by Haynes, of which six died and one recovered. Of these seven, two cases are reported in detail.

Dr. Beck has records of only fifty-one cases of meningitis, but was satisfied that he had seen again as many in consultation or in his clinical experience. Of these fifty-one cases, eighteen had spinal punctures, and septic organisms were recovered from twelve. The diagnosis of diffuse septic meningitis was made in thirty-seven cases of the fifty-one. Of these thirty-seven cases, twenty-eight came to operation either primarily as a nasal accessory sinus, mastoid, or some other local infection, or by way of the exposure of the meninges over the seat nearest the infection; and, finally, the opening of the cisterna magna of the fourteen remaining cases of meningitis, either local, septic or serous of the type. Twelve of these cases recovered without operation, and two died. This number did not include the local meningitis with brain abscess, sinus thrombosis, etc. Of the thirty-seven cases of diffuse septic meningitis, whether operated on or not, only three recovered.

Of the sixteen cases of extradural abscesses, eleven were found at the time of operating for mastoid, sinus thrombosis and frontal sinus disease. Twelve of these sixteen cases recovered after operation. Of the four fatal cases, three were operated on and became complicated by intradural abscess,

meningitis and general sepsis. One case diagnosed refused operation, but a postmortem performed revealed a large extradural abscess in the cerebellar region.

Of nineteen cases of intradural abscess, two recovered. Both of these were in the temporosphenoidal area, and the operation was by way of the mastoid tegmen route. In neither case could there be any microorganism recovered from the pus of the abscess, either in smear or culture. In one case the abscess followed a rapidly destructive mastoiditis in an influenza infection, and the second in the seventh week of a scarlet fever otitis media, in a child aged three years. Of the seventeen remaining cases, ten came to operation. Six were in the cerebellar region, two frontoparietal, and two temporosphenoidal. The seven cases either refused operation or were too far advanced to be submitted to the operation.

Of eight cases of brain tumor, there was made a correct localization diagnosis in five. This number does not include gummata. The pathologic types were cyst, osteoma or exostosis, fibrosarcoma, glioma. The locations were two in the motor area, one occipital (supratentorially), one pontine cerebellar angle, one frontoparietal, one at base of frontal lobe. Five were operated on, with a mortality of 75 per cent. In not one instance did the radiogram reveal the tumor. Spinal punctures were made in seven of the eight cases, and only in one was there any increase in pressure, and in all was a negative Noguchi globulin or Nonne test present.

The cases of brain tumor which Dr. Beck observed were several cases of acromegaly, and three other cases diagnosed only at operation or postmortem. The previously cited case, erroneously diagnosed as cerebral tumor, as well as the two following supposed nasopharyngeal fibrosarcoma cases, illustrated the necessity of refining our diagnostic methods.

His experience with cases of intracranial hemorrhage had taught him that it was very easy to diagnose them, and that early operation gave most satisfactory results, both in fractures aside from the basal and spontaneous subcortical hemorrhage. The use of urotropin in large doses, as much as one hundred and fifty grains a day, had undoubtedly aided in the treatment of nasal fractures.

He cited one case of intractable tic douloureux in which the ganglion was completely removed, but the patient did not receive the expected relief.

DISCUSSION.

Dr. M. Herzog said that it was not at all astonishing that a definite diagnosis could not be made in the case reported by Dr. Beck. The postmortem clearly showed that it must have been almost impossible to locate the tumor. The most common tumors of the brain are gliomata; next in order, sarcoma and endothelioma; the last perhaps still more common than sarcoma. He has had considerable experience in the examination of glioma—probably having seen from twelve to twenty cases. He has never seen one situated as the one reported. As a rule, gliomata develop somewhere in the substance of the brain, and in spite of the fact that it is really a benign tumor, it is infiltrating in character, so it is generally not well differentiated from the brain tissues, the matrix in which it grows, but infiltrates it more or less diffusely. There are, however, some gliomata of the brain which are free tumors, in a certain sense, and those are neoplasma which arise in the ventricles, i. e., from the base or roof of the ventricle, and they project into the ventricular lumen. They are, as a rule, sitting with the broad base on the matrix. It is generally assumed that a glioma originates from an embryologic inclusion, and in some particular cases these neoplasms show an embryonic origin in their structures. The whole central nervous system, as you know, is developed in the following manner: The blastodermic disc becomes folded in, forming an open groove, which then closes and so forms the neural canal, lined by ectodermal epithelial cells, and from these, which in embryologic development have become cut off from the surface, the whole central nervous system is developed. There are quite a few gliomata which in their interior show glandular structures which are lined by epithelial ependymal cells. He has never seen any glioma which projected as a free tumor from the floor of the ventricle into the subdural space, covered by pia, until he saw this case. He supposed that it developed from an embryologic inclusion situated near the floor of the ventricle, and that for some reason or other did not, as is common, develop upwards to the cavity of the ventricle, but developed downwards, and then, in consequence of constriction, formed a slender pedicle, which was so thin that when trying to deliver the tumor at the postmortem examination it broke. The pedicle was no larger in diameter than

a thin thread, and contained vessels which were continuous with the vessels in the ventricle. In turning the brain and examining the floor of the lateral ventricle, he found that the tissue was softened to the touch, and that he had torn through it, showing that the tumor had caused softening of the brain; a change similar to what is often found in dementia paralytica, and this change produced symptoms which led the neurologists to make a diagnosis of general paresis. Some consultants thought that the symptoms developed on a syphilitic basis. The Wassermann which Dr. Herzog made was negative. The examination of the cerebrospinal fluid was also negative.

The clinical symptoms could at least partially be explained by the situation of the tumor. It pressed upon the pons and the medulla, and also upon the lobe of the cerebrum. Dr. Herzog was not present at the last moments of the patient's life, but he understood that there suddenly developed a dyspnea, a condition of passive congestion over the whole surface. The heart kept on beating, but respiration stopped. The respiratory center was undoubtedly compressed upon.

On histologic examination, syphilis (gumma) could, of course, be excluded. Sarcomata sometimes spring from the choroid plexus, or from the tissues lining it, and from the pia arachnoid. These tumors also may develop almost anywhere in the brain.

The speaker made the postmortem examination about three hours after death, at night. The assistant took the brain and tumor and put it in Kaiserling No. 1 instead of formalin. Kaiserling No. 1 is unfit for preserving brain tissue for studying later microscopically to the best advantage. So he could not use special stains, such as Weigert's stain for glia fibers. The tumor, histologically, was composed of cells which were either round or oval. It was very vascular. It had comparatively large vessels, but these were not as irregular and thin-walled as often seen in a sarcoma. A very characteristic feature was that the cells were arranged around the vessels and appeared to spring from around the vessel walls. This is particularly characteristic for gliomatous neoplasms. In this tumor, in spite of the fact that he had not been able so far to demonstrate the characteristic glia fibers, by excluding all other tumors, it could be said to be a glioma of very un-

usual situation. It was a very unique and peculiar tumor—of unique development because it projected not into the ventricle, but to the outside of it, and only connected with its matrix by a very slender pedicle.

Dr. J. Holinger said that he had spoken at the meeting of the International Otological Association in Boston about the diagnosis of brain tumors which have caused absolutely no other symptoms except those from the ears. His attention was first drawn to this subject by a patient with brain tumor who told him that the trouble in the ear preceded by years any other symptom. That fact led him to make a diagnosis and propose operation to a patient who, eleven and a half months later, was operated on for the very thing that he had diagnosed from the functional tests so long before.

The third case that he saw was a patient with a gumma, who gave exactly the same response to the functional tests, but no other symptoms. He got well, of course, after a few injections of mercury.

The fourth patient was a lady whom he had the chance to watch for several months, who was operated on two years ago, after a prominent neurologist in this city said, only a week or two before the operation, that he did not dare make a diagnosis of brain tumor on the other evidences. Still the speaker stuck to his diagnosis, and the tumor was found, although it could not be removed. The patient is still living, and although several symptoms are developing which might suggest a second operation, the scar in the skull is still pulsating, showing that the intracranial pressure is not so very high, and therefore he would not yet insist upon another operation.

With reference to the case of Dr. Pierce, of meningitis, the speaker reported a case which was cured, in the *Zeitschrift für Ohrenheilkunde*, some time ago. The conditions in that case were in every way favorable, and he had an opportunity seven months later to verify the diagnosis at the postmortem, as the woman died from another attack of meningitis after otitis media. He could distinguish the old changes and the new ones, side by side, and could see why recovery was not possible the second time.

He has at present a case of meningitis in a young lady of seventeen, who had distinct symptoms of nasal trouble. The

X-ray showed cloudiness of the ethmoidal and frontal sinuses, on one side. That cleared up. She was up and around for several weeks. Suddenly she became unconscious and remained so for twenty-four hours, when the speaker saw her, and at once had a lumbar puncture made. The fluid was under high pressure and clear. Six or seven years previously that young lady had had a tubercular affection of the external canal and middle ear of the same side, which he had operated on, as a typical mastoid. Therefore, the possibility of a brain abscess was clear. So he suggested trephining in the temporal lobe. He made an opening in the bone the size of a half dollar. There was absolutely no pulsation of the dura. He inserted a very slender knife and found nothing but clear fluid. He explored very carefully in different directions, but while operating the pulse rose from fifty-two to sixty-five and seventy, and she clearly improved. The next morning she was clear enough mentally to recognize her surroundings. In the course of time she recovered sufficiently so that she is walking around now and doing well in every way. She does not complain of headache or any other symptom except a slight weakness in the right leg and right arm. His diagnosis in this case is that it was a case of tubercular meningitis of the base, one of the few cases that recover. Whether she will get entirely strong or not, he does not know; at present, however, she has the appearance of going to complete recovery.

Dr. John A. Cavanaugh said it has been his good fortune to have seen two or three of the cases mentioned by Dr. Beck, and what impresses him most was a complaint of headache by the patients. The other signs were practically negative. One of the cases, a young man, Dr. Cavanaugh had seen while Dr. Beck was out of the city. Spinal punctures were absolutely negative. The only thing of any importance whatever was the headache. At times the patient was a little irritable. The only thing that suggested the brain complication was the fact that a mastoid had been done, and the probabilities were that there was an involvement in the brain secondary to the operation.

Dr. Cavanaugh examined the patient and thought it best to expose the dura in the region of the mastoid and explore, but while preparations were being made for the operation the patient became cyanotic and died.

In the case of the elderly man, reported by Dr. Beck, spinal puncture just before operation was negative. The only symptom of importance was the headache.

DR. L. W. DEAN, of Iowa City, Iowa, said that the only treatment that had been of any value in his experience, so far as diffuse septic meningitis was concerned, was the prophylactic treatment. He has done a subdural drainage on some eight or nine cases. They all died. He questions the advisability of being guided by the rule laid down in Neuman's table, which states that in individuals with loss of hearing, functionating semicircular canals, no fistula symptom, and chronic otorrhea, only a radical operation, not a labyrinthectomy or labyrinthine drainage, should be performed. Following his interpretation of this rle, he did not operate upon the labyrinth in the following case. The patient had a discharging ear for fifteen years. Caloric test was positive. There was no hearing. Following the rule of the Vienna school, he did only a radical operation. Keeping in mind that the Vienna school suggested that in such cases a very careful examination should be made to see if there was any evidence on the inner wall of the tympanum of involvement of the labyrinth, he made a very careful examination. No necrosis or evidence of fistula was discovered. The head of the stapes was in good condition.

Following the radical operation, nothing of moment occurred until two weeks after, when the patient developed a slight facial paresis and a temperature of about 100.° The next day he had projectile vomiting, paresis of the facial, rigidity of the muscles of the back of the neck, temperature of 103,° and extreme headache. Spinal puncture showed a very turbid, white fluid under great pressure. It contained 97 per cent polynuclear cells. No bacteria could be found either in coverglass preparations or by making cultures on Loeffler's blood serum or agar. Fehling's solution was not reduced. Noguchi globulin test was positive. In his judgment this case was not at this time a diffuse septic meningitis, but was a circumscribed meningitis, with a general serous cerebromeningitis. A labyrinthectomy was performed. Before operation the caloric test was positive. There was no pus either in the semicircular canals or in the cochlea. As soon as the internal auditory canal was penetrated, half a thimbleful of pure

white pus escaped. This was followed by two or three ounces of turbid cerebrospinal fluid. This patient made apparently a most beautiful recovery. His headaches left him. The rigidity of the muscles of the back of the neck disappeared. The temperature became normal, and he was able to sit up in bed. About ten days after the labyrinth operation, with drainage of the meningeal spaces, rigidity of the muscles of the neck became suddenly manifest again. Temperature was elevated. Spinal puncture gave the same findings as in the first case, except that in addition streptococci were found present. This case was now considered one of diffuse septic cerebromeningitis, and the condition practically hopeless. The patient died. The temporal bone is being decalcified and has not yet been examined.

If he had a similar case now he would not be guided by the Vienna school and be content with a radical mastoid operation, but would perform a labyrinthectomy.

Dr. S. A. Friedberg spoke of the difficulties in differentiating various intracranial lesions outside of sinus thrombosis or distinct meningitis. With Dr. Loeb and Dr. Beck, he had that day seen a case at the County Hospital—a boy of about twelve, with a history of suppuration of the right ear for five or six years. He had scarlet fever up to three weeks ago, from which he had made a recovery, and examination of the ear showed a central perforation—some purulent discharge—no mastoid tenderness. The boy was brought to the hospital two day before, at which time the following history was elicited: He had been having severe frontal pain for four or five days—had vomited once two days before admission, and again the night before coming to the hospital. Pulse on admission was around 64; temperature 99.° That was about the highest the temperature had gone. The speaker, with Drs. Loeb and Beck, examined him and found that he had a peculiar type of respiration—socalled air hunger, every few minutes yawning, as if getting insufficient air. Pulse still kept down; temperature not above 99.° Complained of headache. Dozed a great deal. When awakened he responded readily to questions. He spoke of having some double vision, but this had disappeared. The speaker thought there might be a slight ptosis of the right eye, but this was not definite. There was no evidence of any other involvement of the cranial cavity.

He had nystagmus—both horizontal and rotary—which was spontaneous to either side, perhaps slightly more pronounced on being directed to the opposite side. In testing with the cold water the nystagmus was produced for about twenty to twenty-five seconds. Spinal puncture showed the fluid not under pressure, clear, no bacterial content, and reduced with sugar to a certain amount. No rigidity of neck; no distinct meningitis. X-ray negative. Dizziness was complained of. In testing for ataxia, he fell to either side. The hearing, outside of the middle ear process, was pretty fair. There was one other sign elicited—diadochokinesia. On the whole, the patient seemed rather apathetic.

The speaker thought meningitis and labyrinth trouble could be excluded from the diagnosis. The question was as to the localization of the trouble. He had practically no focal symptoms. Of course, the evidence was there of pressure, as manifested by the pulse and the spinal puncture. Whether it was cerebral or cerebellar, he had not determined as yet. He simply brought this up as pointing to the difficulties encountered in localizing these intracranial conditions at times.

Dr. H. W. Loeb, of St. Louis, said that as this seemed to be in the nature of an experience meeting, he would briefly cite a case of a child, perfectly healthy except for a running ear, which had, from the history, been entirely healed for several months, who was suddenly taken with acute otitis media. The mother said that the child had put a piece of paper in his nose. This history, however, was not elicited until some four or five days after being seen. He found a large piece of paper in the right side of the nose, and was quite sure that the acute exacerbation had its inception in that. The child was taken almost immediately with symptoms which were taken to be those of meningitis—he had a positive Kernig and other symptoms that go with meningitis. Mastoid opened up and the middle fossa at the same time, but nothing was found. The symptoms continued, though somewhat modified, the spasms having been replaced by twitchings, the child going into a comatose condition. So he decided to operate in the posterior fossa. In order to get plenty of room, operation was made through the occipital bone, posterior to the mastoid wound, and not through the mastoid opening. A large opening was made, a large button taken out, incision made into

the cerebellum and no abscess found, although abscess of the cerebellum had been diagnosticated. Postmortem examination showed that there was a very small abscess in the cerebellum, which had not been reached by the operation. Hence he said it was a case of successful diagnosis and an unsuccessful result, because he did not happen to strike the pus. The postmortem showed no meningitis present.

DR. G. W. BOOT reported a case, seen several years ago, of a young man who had a chronic suppurative otitis media for several years and who was taken with symptoms pointing to brain abscess. He was operated on by the speaker, who cleaned out the mastoid, opened the dura and sinus and explored the cerebellum for an abscess. This patient died after several days, and the postmortem showed that he had a cerebellar abscess, which the speaker had only missed reaching by a quarter of an inch.

He thinks we should always remember, in such cases, to look for the abscess not far from the internal auditory meatus. It is apt to be there instead of farther back.

Another case was referred to him for diagnosis of the cause of headache. In examining the young woman he found that she had very marked choked disc. He took her to see Dr. Wilder, who confirmed that, and advised him to take her to a neurologist. This was done. The neurologist thought she had a brain tumor but could not locate it., He returned the patient to the first doctor, who operated that afternoon or the next day, intending to do merely a decompressive operation. There was considerable hemorrhage at the time from the diploe. No tumor was found and the patient died the same night. The brain was removed and hardened and later sectioned, but no tumor was found. However, there was a hemorrhage occupying a space about half an inch in diameter in the cerebellum.

In this connection he thinks it is imperative to remember that the symptoms of brain tumor may be imitated by a marked anemia, and in every case where we wish to exclude the things which might be confounded with brain tumor we should always remember the possibility that the same symptoms may be set up by anemia.

Another point in connection with brain tumors is that where brain tumor is suspected, we should be very careful about lum-

bar puncture, because when the pressure is removed the tumor may force the medulla down towards the foramen magnum and so compress it as to cause sudden death.

He reported the case of a girl, about ten or twelve years of age, who had had a chronic suppuration from the ear for a number of years. He was called to see her because of peculiar brain symptoms, and suspected that she had a brain abscess. She was sent to the hospital for operation on the mastoid, but while preparing for operation, and before she was anesthetized, she died. In that case he always believed that the girl had an abscess which ruptured into the lateral ventricle.

Dr. J. Gordon Wilson said that the paper and discussion had been suggestive as to the lines along which otology is developing. To limit otology to the temporal bone is to take a narrow view of its sphere of action. As the ophthalmologist is called to give his opinion in regard to cranial tumors, so the time would come when the otologist would be asked to assist in locating and diagnosing lesions of the brain. Already we see what has bene done by the application of the caloric and rotation tests. Further, as a considerable proportion of brain abscesses come either directly or indirectly within the field of the otologist or rhinologist, we are compelled to have at least some knowledge of cranial symptoms.

Another thing which has made this meeting most interesting is the careful and painstaking history of an obscure case of brain tumor. It is too often the case that in clinical records of brain tumors there is something essential wanting. There is no place in the body where the demand is greater for a full and careful report of symptoms, not only in the case recorded, but also for future reference. He wished to compliment Dr. Beck on the care with which this history was recorded.

In the paper of Dr. Pierce he recorded the microorganism he found. Dr. Wilson has always felt that we should be a little more particular in ear cases to ascertain the organism we are dealing with. By this recognition we can aid the prognosis and at times the treatment. Thus in time we shall get to recognize better the more dangerous organisms. The most fatal cases of meningitis he has seen have been associated with the streptococcus capsulatus.

Just one other point, in regard to the case referred to by Dr. Friedberg. Dr. Wilson has always felt that if in any case the nystagmus is directed to the diseased side, the great probability is that we are not dealing with a case of labyrinthine disease. One could imagine a case of labyrinth disease in which there is hyperexcitability of the semicircular canals, but they must be very rare.

DR. BECK, in closing the discussion, said that the question of diagnosis by the aid of the X-ray in intracranial conditions has been a terrible disappointment to him. He has yet to find one case in which he could say that he had located a certain thing, like an abscess or tumor, by the X-ray, which subsequent operation or postmortem could show. Perhaps it was in the interpretations. Maybe in the technic. Of course, in cases of fracture, the X-ray was very useful, but in deep-seated intracranial lesions he has not been able to show its real value.

The speaker reported the case of a man with tumor at the base of the skull, in which case neurologists had made a diagnosis of hysteria. In looking up the literature he found that Cushing reports two such cases of pain, even after gasserectomy, which was performed in his case.

There was one case of encephalocele which was very interesting. He made serial sections of the temporal bone in this case. Professor Denker and others, who looked at them at the last Congress in Boston, agreed with Dr. Beck that there was an underdevelopment of the finger structures in the internal ear. So a point to consider in such cases is that, even if the encephalocele is cured, the patient's hearing will perhaps never be of much value. The investigation of such cases that have lived, either with or without operation, would add a great deal to the subject.

Regarding the case Dr. Cavanaugh spoke of, in which he said that the reaction in the spinal fluid was not the same as the normal copper reducing reaction, that was not the only symptom. The main symptom present was a purulent fluid—a fluid that contained microorganisms from the very beginning, and that was the reason the speaker operated.

DR. CAVANAUGH asked if it reduced to copper?

DR. BECK replied that it did not. He operated because the fluid was infected. With regard to these cases, he would urge

men to follow them up and try to get a postmortem, because we can certainly learn more from that than anything else. He would say openly that he had been successful in stealing his postmortems over at the Cook County Hospital, because the doctors are not supported in this by the authorities. Permission should be given for postmortems.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of May 27, 1913.

THE PRESIDENT, DR. J. GORDON WILSON, IN THE CHAIR.

Vincent's Angina.

DR. OTTO J. STEIN said that there was nothing unusual in the case of Vincent's angina which he presented in the boy, but he wanted to show the specimens correctly, side by side with the clinical case. These specimens are very profuse in the characteristic organisms—the spirillum and the fusiform bacillus, and in this case he thought he would try the inoculation of the opposite tonsil. He took some of the smear from the left tonsil, which was involved at the time, and after abrasion of the tissues of the right tonsil, the one not involved, he put this secretion underneath. Being anaerobic, the bacteria must be buried. Both tonsils are now ulcerated. (Later: Two weeks after showing this case, both tonsils were removed completely by Sluder technic, with no infection of surrounding tissue.)

In the second case the

Double Abductor Paresis

was rather unusual. He had not seen this man since a month ago, and he presented a typical abductor paresis at the time. The right cord showed an involvement of the internal tensor as well, with serrated edge and shortening of the cord, and a slight dipping inward of the arytenoid ridge, while the left cord showed nothing but involvement of the abductor up to the vocal process. The little chink was present in the back. On attempt to separate by deep inspiration, the cords made a quiver—just an effort and nothing more—there was no abduction whatsoever. Examination for some cause in the neck and chest revealed nothing physical. There was no evidence of anything, and only the X-ray demonstrated the presence of the aneurism which involved the entire arch around to the end of the third division of the arch. Dr. Stein understood,

from what internists told him, that it is not unusual at all to have absolutely no physical manifestation of the aneurism for some time, while the X-ray pictures will show it always before such manifestations develop, as in this particular case, which demonstrates very nicely what the laryngologists can do sometimes in arriving at an early diagnosis.

Demonstration of the Struycken-Schaefer Monochord.

DR. ALFRED LEWY: A number of attempts have been made to construct a practical instrument for testing the hearing for higher tones. Besides the Galton whistle there are the Koenig rods, the Melde stimmplatten and the monochord. This one is the Struycken-Schaefer monochord. It was preceded by the Schulze monochord and others not so practical and less accurate. This instrument, as devised by Struycken and improved by Schaefer, consists of a metal frame 50 cm. long spanned by a piano wire on which there is a sliding block which alters by its position the length of the vibrating string. The number of vibrations, and therefore the pitch, depends, of course, upon the length, weight and tension of the string. The frame is calibrated so that the pitch is known by the position of the movable block. A scale for reading the pitch of the longitudinal vibrations is on the instrument. In the *Laryngoscope* for March, 1913, Dr. Goldstein published with his article a scale for translating the calibrations for transverse vibrations into pitch. Typographical error designates the pitch in capital instead of small case letters. By using both the transverse and longitudinal vibrations the range of the instrument is from about g^1 to beyond the high limit of human perception. As it can be used for bone as well as air conduction, it supplements very well the Bezold set of forks. It has the advantage over the Koenig rods and the Melde stimmplatten in not requiring numerous separate instruments, and over the Galton whistle in testing the bone as well as the air conduction. Besides, it is claimed by Schaefer, and so seems to my ear, that the highest tones are purer and more easily distinguished from the usual adventitious sounds.

It is best tuned by setting the block at 43 and then screwing the tension so that the transverse vibrations correspond to the a 1 fork accompanying. (This fork does not quite correspond

with the Edelmann a^1.) There is a little calibrated weight accompanying the instrument for measuring the tension, but I have not found it practical.

For the lower tones—the transverse vibrations—the handle is rested on a table to act as a resonator (otherwise the tone is too thin), and the wire is struck by the cork hammer or rubbed with the little violin bow. The highest tones—longitudinal vibrations—are obtained by stroking the wire longitudinally with a felt pad wet with turpentine, benzol, carbon tetrachlorid or alcohol. A metal flask so equipped accompanies the instrument. The wire should not be fingered, on account of rusting. To test bone conduction the bone button is attached to the tip of the instrument and placed against the mastoid process while the tones are elicited.

While this instrument can be used for testing all tones from g^1 upward, its practical application is most important in the testing of the high tone limit, which with this instrument is d^7, 18,581 d. v. for the trained ear. For the Galton whistle it is said by Edelmann to be f^7, 22,097 d. v. In a number of patients in whom I made comparisons, the monochord and the Galton whistle seemed equally accurate in detecting slight losses of the upper tone limit. For the ordinary ear c^7, 16,554 d. v., may be considered a normal standard. Wolff, of Berlin, after testing forty patients with this instrument, came to the conclusion that characteristic for nerve deafness is a greater loss of perception for high tones by air than by bone conduction, the discrepancy in some cases being quite marked. That is, bone conduction is better than air conduction for high tones, the reveres being true for lower tones. This also occurs, he finds, in marked effusions of fluid in the middle ear or external canal. Normally, and in pure conduction deafness, except when effusions are present, the hearing by bone and air conduction for high tones is about the same. You will recall that with the tuning fork, a 1 for instance, the loss by bone conduction is the more prominent in nerve deafness. Struycken explains this by saying that the vibrations of the handle of the tuning fork are only about one one-hundredth the intensity of the tines. No doubt the pitch is also a factor.

My own investigations so far are limited to twenty-three cases; three of spongification, five nerve degeneration, five otitis media chronica, six mixed, two chronic suppuration, and

two of doubtful diagnosis. Two of the cases of nerve deafness are believed to be due to tobacco. So far my findings agree with those of Wolff, i. e., bone conduction is better than air conduction for high tones in nerve deafness. Permit me to express here my opinion that the condition of the high tone limit is of value in prognosis, and that any considerable loss of hearing for high tones, except in hysteria, is of bad prognostic import, regardless of the original cause of the trouble.

My thanks are due Dr. Pierce for the opportunity of making these investigations in his clinic.

DISCUSSION.

DR. ROBERT H. SONNENSCHEIN said as to the difference in the appreciation of high tones by bone and air conduction, that he believes, as stated by physicists, and as mentioned by Dr. Lewy, very high tones are better heard by bone conduction than by air conduction, and the striking difference between the amplitude of the prongs and the stem of the fork accounts for that. If the amplitude is the same, high tones are heard equally well by both, but in the small c 4 and 5, the amplitude of vibrations of the stem of the fork is only one one-hundredth that of the prongs, which accounts for this discrepancy.

So far as he can see, with the use of the instrument demonstrated by Dr. Lewy, with which he has had only slight experience, it is very difficult in a great many cases, when you get up as high as c_6 and c_7 to get an appreciable tone constantly. Sometimes when you rub it you get a squeak here and there, but, as a rule, you cannot get a consistent tone. Of course, when you get down to small c 4 and 5, you can, but you can get that with a good tuning fork. So thus far he cannot see any very great advantage as compared with a small c_5 fork.

DR. GEORGE E. SHAMBAUGH said that he is coming to appreciate more and more that the testing of the upper tone limit is one of the most valuable tests that we have in diagnosis as well as prognosis. A c_4 tuning fork does not suffice for this test—except in advanced stages of labyrinth deafness, but where one gets an appreciable loss of hearing for the c_4 tuning fork, it may be dispensed with, because the other tests show the condition.

The testing of the upper tone limit is especially valuable in the study of cases of advanced long standing middle ear deafness. In many of these there begins to develop, sooner or later, a degeneration in the labyrinth—labyrinth deafness, which one cannot detect with the Weber and Rinné, or the Schwabach tests, because these tests will continue to give the characteristic reactions of middle ear deafness. The quickest way in which one can detect beginning labyrinth degenerations is by testing the upper tone limit. A recognizable defect in the upper tone limit is of decided significance prognostically.

DR. J. HOLINGER said that Burkhardt-Merian, of Basle, was the first to recognize the diagnostic importance of the upper tone limit and used it systematically. He employed a whistle which he perfected in all details, calling it "Galton whistle." The original Galton whistle had simply the movable piston, and was not made for the highest sounds. Burkhardt-Merian determined the form of his mouth and its relation to the bore of the whistle. He introduced the screw and gauge on the piston. It ought to be called "Burkhardt's whistle." Some of these Galton whistles have done pretty good service. Others were not executed so carefully and accurately; therefore, the results were not uniform. Later on Edelmann knew that the upper limit of his series was the most unsatisfactory part. He tried to gauge his whistles by means of Schwendt's dust figures. But even in 1899, when Dr. Holinger got his set of tuning forks, there was a rumor that the Galton whistle was not scientifically accurate. It was said that in the highest pitch a turn of the screw of the piston means a rise in pitch of half an octave; in the lower region of the whistle only one or two tones. Therefore, the original Edelmann's whistle could not be considered a "scientific" instrument. It was improved later on. In 1909, when abroad, the speaker saw the monochord in use very extensively, but it was an entirely different instrument from the one demonstrated by Dr. Lewy. He had the impression then that it was not an instrument to be used in the office every day, because it was unhandy. The instrument shown by the essayist is carefully gauged and is much more handy. This is necessary in an instrument of that sort. Dr. Holinger has not paid very much attention to the determination of the higher tone limit in the last few years, but now that we have a workable instrument, he intends to take

a greater interest in this part of the functional tests. The great many improvements that Edelmann made of late in his whistle made the whistle also a workable instrument, but still every once in a while we read criticisms from scientists who are in a position to really verify and prove their contentions.

DR. WILLIAM L. BALLENGER has had no experience with this instrument, although he has owned one for years. He did not understand just how to use it until Dr. Goldstein's article came out last March. He has had great difficulty with the Edelmann-Galton whistle. The instrument he has was made in 1903. It can be tuned in different pitches. That is, you cannot set it and then go up all the pitches by turning the main apparatus, but you must turn a tuning apparatus at the other end of the instrument, and he finds great difficulty in getting the same intensity of pitch, and hence he has felt that it was not a very useful instrument in his hands. But with this instrument we can have a constant pitch and greater accuracy. It is a simple instrument, and he hopes to make use of it in the future.

DR. LEWY, in closing the discussion, said that the practical advantage that the instrument he exhibited had over the Galton whistle is that it assists us in obtaining the bone conduction for high tones as well as the air conduction. The Galton whistle gives us only the air conduction. It may be that through this we will be able to add to our diagnostic and prognostic ability. He simply presented the instrument for what it was worth, and in the interests of the society.

Paper: The Nasal Voice With Reference to Its Bearing on The Practice of Rhinolaryngology.*

BY ELMER L. KENYON, M. D.

DISCUSSION.

DR. J. HOLINGER thinks that there is another phase to the question discussed by Dr. Kenyon. Dr. Kenyon warned against the removal of adenoids under all circumstances, without regard to other organs. This is a timely suggestion. At present the speaker is treating a child whose adenoids had been removed about a year ago by a laryngologist who does not

*See page 1110.

make any pretensions of treating ears. The boy has highly retracted drum membranes, is therefore hard of hearing, and has a decided defect of speech, consisting of an apparent rhinolalia. The speaker tried to study the case, and found that in pronouncing the patient's own name, Norman, the defect was most evident. He pronounces it Noonan. The consonants "r" and "m" are of very low pitch and not heard well, and therefore are not pronounced correctly by the otherwise bright boy. His rhinolalia is nothing but the consequence of lack of hearing. The ear was treated three or four times, and the mother was instructed to watch the boy. It was surprising how quickly he improved; he speaks much better now. Dr. Holinger thinks it is often not so much a question of instruction as a question of correcting the condition in the ear. That was his contention for many years as to the adenoid operation in the hands of men who do not treat the ear, and who do not pay any attention to the ear after the operation. They will overlook a retraction of the drum membrane, and the consequence is that they practically have not benefited the patient very much. Considering the experience with this boy's case, might there not be a possibility to explain rationally the case of Professor Gutzmann as that of a patient who did not hear well as a child and therefore missed the right pronunciation?

DR. JOSEPH C. BECK was very much interested in the paper, and it reminded him of some cases that he had had of speech defects. One case he showed before the society several years ago, of rhinolalia aperta, in a case of congenital short palate, which, through training, before he had done anything, refused to respond. He subsequently injected paraffin behind the mucous membrane in the region where one would expect this palate would meet the posterior margin of the soft palate, and immediately after the injection this patient began to speak more clearly and distinctly, and the result was very satisfactory indeed. There was an interesting fact about this patient, in that was she was a young woman then of twenty-five, and did not know that she had this defect. The doctor wanted a record of her speech before he did anything for her, and so he thought of Dr. Brown, of Milwaukee, who had recorded his cases by phonograph, so he had her talk into a machine, and when she heard the record she was completely paralyzed and

fainted away, for she did not know she had spoken that way.

When Dr. Kenyon spoke of educating the patients so that they know how they speak, it is perhaps by the aid of that nasal ear, and Dr. Beck thinks that perhaps in that way they may be able to hear how they speak. Dr. Patrick spoke to him once of a condition, the German name for which he has forgotten, in which the patient does not understand or cannot hear his own voice. In fact, the speaker thinks it is quite common, and that most of us are surprised at hearing our own voices in the phonograph—they sound different.

He was reminded of another case following removal of adenoids, in which the patient developed a distinct rhinolalia aperta, and he thought that he had perhaps traumatized the palate. There was perfect action in the palate. The condition did not improve for some time. He examined the patient very carefully, and found a very soft portion of the hard palate, and in taking a dental radiogram he found a marked deficiency of bone in the hard palate—a congenital deficiency. This voice defect was not present while the adenoids were present, but when he had removed the adenoids—which was not done for any other purpose than a distinct systemic condition—this speech defect occurred.

Another point: The doctor said it is difficult to examine the palate. Dr. Beck has examined the palate, and you can see the action of the palate during speech and see what condition it is in.

Dr. Alfred Lewy said that one idea that impressed him after hearing Dr. Kenyon's paper was the great neglect of the entire subject of speech defects by the medical profession in this country. Most of the treatment of speech defects is done by people who advertise to cure stammering, and use bizarre methods, such as causing the patient to sing the words or to go through certain peculiar motions, and so forth. In the public schools in Chicago some effort has been made to train special teachers in the correction of defective speech, but altogether in this country the subject has been very largely in the hands of charlatans, and he thinks it is largely the fault of the medical profession, and that we need such papers as that of Dr. Kenyon to show us the importance of the subject and the possibilities that lie therein.

Dr. Kenyon, in closing the discussion, congratulated Dr.

Holinger on his diagnosis in the patient whose hearing was impaired. It is often not an easy diagnosis to make. Many a child talks badly because he does not hear correctly. Many a child does not talk at all because he cannot hear exactly, and he may not be more than moderately deaf.

Paraffin injection in palatal cleft is, of course, a valuable procedure. He referred to it merely to point out that adenoid tissue serves exactly the same purpose as the paraffin—it helps to prevent the air from going upwards into the nasopharynx, when the impaired palate does not sufficiently perform that function.

It would seem as if the nasopharyngoscope passed through the nose into the nasopharynx might aid in diagnosis in difficult cases of imperfect palatal movement, by enabling one to watch the movements of the palate; but diagnosis is usually possible by the much more simple methods described.

So far as the patients not recognizing their own voices is concerned, that is very true. None of us know our own voices, and patients with the most apparent defects of speech —if they be children and have never been told about their incorrectness of speech—believe that they talk as well as anybody, and they have to be told in order to know that they have a defect of speech.

The Value of Skiagraphic Plates in the Study of the Diseases of the Nasal Accessory Sinuses and Mastoid Process.

DR. W. L. BALLENGER presented a number of very interesting and instructive photographs of skiagraphic plates illustrating the value of skiagraphic plates in the study of the diseases of the nasal accessory sinuses and mastoid process. He presented two plates which showed a single ethmoid cell involved in each instance, and brought out the fact that in such cases the operation for the drainage and exenteration of the diseased cell could be done with a minimum amount of surgical trauma. In one of these cases the patient had been repeatedly operated by a competent specialist, but without relieving the suppurative symptoms. Dr. Ballenger, by means of the plate, located a single ethmoid cell, on which he operated, with entire relief to the suppurative process. In the other plates showing a single cell involved it had been diagnosticated as a case of cerebrohydrorrhea because of the pro-

fuse watery discharge from the patient's nose. The plate, however, shows one cell located under the right frontal sinus to be involved. The interesting points concerning this case were the possibility of locating a single infected ethmoid cell, which was giving rise to a nasal hydrorrhea. There was no purulent discharge in this case, but in spite of this the cell was distinctly cloudy, as much so as in suppurative disease. It also cleared up the diagnosis. He also showed plates showing the osteoporotic destruction of the inner plate of the frontal bone from luetic disease. Also plates contrasting a healthy mastoid process with a diseased process.

Plates showing extensive fractures of the skull were also shown, in which the patient, while riding in an automobile, collided with a telegraph pole and was rendered unconscious for three or four days. On recovering he was found to have nystagmus to the opposite side, nausea and vomiting. On examination by Dr. Ballenger, two months later, the nystagmus, nausea, etc., had disappeared, but he still suffered from disturbance of equilibrium, and was totally deaf in the right ear. The plates show a large arch fracture extending from the outer angle of the left orbit to the inner angle of the right orbit, and a Y-shaped fracture extending through the temporal bone on a level with the cochlea and vestibular apparatus.

DISCUSSION.

DR. R. C. LYNCH, of New Orleans, said that he had been using X-ray plates in his work in New Orleans for the last six or eight months, and has found that they help very materially in every branch of the work, especially in the diagnosis and proper interpretation of sinus suppurations. One case of mastoiditis that gave him a good deal of concern, because of the very chronic character of the inflammation and of so few clinical symptoms that accompanied the condition, was really treated successfully on account of the use of the radiogram. This patient had the usual onset in an acute otitis; the drum ruptured. He was an ignorant man and neglected it for some time, and then began to suffer with headache, and the headache was the only symptom that he gave of any disturbance. His headache increased in intensity until he came to the clinic for observation or for some relief. The X-ray pictures showed the normal side to have a very extensive cellular mastoid, the

cells extending well forward into the zygoma, very much higher than usual, over the roof of the tympanic cavity and far back, and especially the tip was very large and cellular. On the diseased side it showed the process to extend even into the diploe, and he was rather suspicious of a low grade osteomyelitis developing in this case in consequence, or, rather, as a result, of the extreme degree or the great amount of involvement of the bone, as shown by the X-ray. In doing the operation he extended it well beyond the limits that he would have, had he not had the X-ray picture at his side. This man recovered without any disturbance. Dr. Lynch believes that if he had not had a radiograph at his disposal, and the measurements taken carefully before operation by the radiologist, the extent of the operation would not have covered the diseased processes, and the consequence would have been that this patient would not have recovered in the very nice and proper way that he did under this treatment. So that he believes that the X-ray is very valuable. We are really beginning to look upon it now with far more favor than we ever have, and he believes that it is opening up more and more new fields for diagnosis. He had the pleasure of discussing this phase of this subject with Dr. Ingersoll, who has been doing some work on the internal ear. He feels, Dr. Lynch believes, that he can outline the semicircular canals, though rather indefinitely, by X-ray. It is not at all impossible, the speaker imagines, to have the X-ray help us materially in the diagnosis of certain conditions of the internal ear. He made that statement, not as a definite fact at all, but rather as a thought that came to him at the time, that it may be that we may devise some method in the future of helping us along this line. Certainly, if the X-ray can be of service in these conditions, it will be a source of pleasure and benefit to us.

Dr. Joseph C. Beck said that it was unfortunate that Dr. Ballenger could not have shown the plates instead of the photographs. It has been, by radiologists all over the world, a prohibitive thing to demonstrate facts by photographs, and he was partially to blame for this exhibition to-night by photographs, because he promised Dr. Ballenger that he would bring down these frames for showing the plates. So he wished to apologize.

It was certainly a pleasure to him to hear men express them-

selves in such a way regarding the use of the X-ray. He was and is very enthusiastic over it, but still he might say at this time that he is losing a bit of this great enthusiasm.

The fine points that Dr. Ballenger brought out, of localizing one single cell, he would take issue with, especially in a single radiogram, for what do we know by looking at one plane from one point of view how many cells are involved. With stereoscopic pictures, however, we can tell better how many cells are involved. The speaker would not like to go on record as saying he could make this diagnosis.

Dr. Lynch brought up the point of radiography of the semicircular canals. Dr. Beck has tried to do this, and in not a single instance in the living could he determine anything further than the internal auditory meatus, which he for a long time took to be the middle ear. If you examine with the X-ray and see a distinct outline of the middle ear, that is not the middle ear, but is the internal auditory meatus, and is diagnostic of tumors of the auditory nerve.

When we speak of taking X-ray pictures of these bony structures through bone that is as dense as that, it does not seem possible in the present stage of radiography. Stereoradiography of the sinuses and mastoid is the coming thing.

Another thing: To leave the interpretation of the condition that you are dealing with to a radiologist is a mistake. Leave him out entirely. Just see that he takes it correctly, and make your own interpretation of the print.

DR. BALLENGER, in closing the discussion, said that Dr. Beck was entirely mistaken about the impossibility of showing a single cell except by the stereoscopic method. If the print is taken from the corner of the head, as it were, through the orbit, and not the front view, in this way you get a perspective view of the cells back to the sphenoid, and as far forward as the frontal and anterior ethmoid. You can see the cells one by one from the front to the rear, and then, if you will take the opposite side, you will also see that same cell from the opposite side, but from a different angle, and in that way, by taking two plates of the cell, one cannot be misled; and if the cell is one that can be penetrated with a probe, if it happens to be so located, you can place it accurately, and only remove that one cell, as was done in one of the cases reported.

In the second case reported he removed two or three cells.

The point he wanted to make was simply that it is possible to do this without stereoscopic plates. In taking the pictures as suggested, one plate shows absolutely every sinus of one side of the head, and the second all the cells on the opposite side, and he knows of no possibility of being confused by this method.

Paper: Caisson Workers' Deafness: A Preliminary Report.*

By George W. Boot, M. D.

DISCUSSION.

Dr. Peter Bassoe (by invitation) said he was glad that Dr. Boot had reported this work to the society, because otologists can do a great deal toward clearing up various points about caisson disease, and also there are things in caisson disease that come as near animal experiments on a human being as anything can be, and if properly carried out at the proper time may help to clear up many points in otology.

We must bear in mind that the man who works in compressed air is subject to trouble when he goes into the compressed aid and when he comes out again. So far as ear conditions are concerned, the most common ones, such as "blocking," are brought about during compression, and the ear is the only organ affected by the state of compression. Of course, what happens when the air pressure is rapidly rising is that the air is all the time being compressed, there is more oxygen and nitrogen taken into the blood, and in order to avoid being "blocked" the middle ear must contain the same kind of air as the outside air, otherwise there will be unbearable pressure on the drum, hence every worker is taught to swallow air while going in, and if he does not do that he invariably gets "blocked." Some of those who have investigated very extensively along this line believe that primarily all of the ear troubles of caisson workers are due to this stage of the compression—that the chronic troubles that follow in the middle ear and internal ear are secondary to this disturbance that occurs during blocking. The work of Dr. Boot has shown that we do deal with a good deal of labyrinth trouble in many of these cases, and it seems to the speaker that he has shown pretty well that the stage of decompression

*See page 1121.

has a good deal to do with it. Practically all of the other forms of caisson disease are due to the liberation of the air from the blood during too rapid decompression. That is the period of danger in caisson workers. Air bubbles are liberated in the joint fluids, cerebrospinal fluid, or spinal cord, and they may become paralyzed. Dr. Bassoe examined about a hundred and eighty men, over half of whom gave a history of trouble coming on after they came out of the caisson, particularly the cases of "blind staggers." They come out, are extremely dizzy, vomit, and often become deaf. It is of interest to figure out what happens in those cases, and whether it is liberation of air in the endolymph or a liberation of air in the auditory nerves—perhaps sometimes in the brain. There is here a possibility of the production of local lesions that might be of great interest to otologists. It has been suggested, for instance, that a local lesion might produce tone islands or other interesting disturbances, but so far as he knows trained otologists have rarely gone to work on these cases. Some work has been done in Vienna, where some very painstaking observations were made. It would seem that otology might profit from a study of this disease, because it is almost comparable to an experiment. You take a man from the normal pressure of fifteen pounds and put him in forty-five or fifty pounds, in so many minutes, and let him out in so many minutes, and then study the conditions present. We have this work going on nearly all the time, with wonderful opportunities for research. In the fatal cases we now know quite well what we are dealing with, namely, air bubbles in the blood. There is sometimes the condition spoken of as "chokes," in which the patient becomes suffocated and cyanotic. He sometimes recovers, but more often dies, and it has been found that the right auricle may contain huge air bubbles.

Dr. J. R. Fletcher thought that the cases referred to by Dr. Boot having the blind staggers are very likely serous labyrinthitis. It seems to him that where the air bubbles are liberated in the cochlea or in the semicircular canals, the damage would be very great to the organ. After mastoid operations we frequently see the production of serous labyrinthitis, and it acts in just the same manner that Dr. Boot mentioned in his paper, and it is not incomprehensible at all

that the increase of pressure should produce a rather prolonged hyperemia, primarily, which would produce what we are justified in calling a serous labyrinthitis. In the cases in which the damage is very great there must be then a large quantity of fibrin in the serum, which becomes organized and does the real damage. So long as there is not much fibrin thrown out the recovery is probable, but sometimes very slow, even extending over years. In operative cases—not caisson workers—it may extend over a long time, but the majority recover.

DR. BOOT, in closing, said that the most characteristic thing in these cases is the degeneration of the auditory nerve, just as you see in deafnes resulting from occupation, boiler makers' deafness, for example—a slow degeneration that goes on, not at the time the men are working, but afterwards, after they have ceased working. This degeneration continues for a considerable length of time, and they lose the upper limit of hearing. That, he thinks, is the most characteristic lesion in these ear cases.

Another point of considerable importance is the fact that the lesion of the vestibular portion of the nerve, whatever it may be, seems to be recovered from completely. There was only one case where he did not get perfect response to the caloric test.

He doubts very much whether the points made by Dr. Wilson had very much to do with the case. When going under compressed air the endolymph would be forced out, and as they came out it would be forced back in again into the endolymph canals.

DR. WILSON said that he thought the perilymph is the part that is first affected in the compression.

DR. BOOT thinks the blocking is purely middle ear trouble, the same condition we see in coming up and down in elevators, and which is relieved by swallowing, and these men are instructed to swallow. The other features he thinks are of the labyrinthine type—changes in the labyrinth.

He had hoped to be able to make some experiments with guinea pigs, in order to get the microscopic appearances of these ears, but as yet has not had an opportunity to do so.

DR. STEIN asked whether any of the cases had died and postmortems been performed, to which Dr. Boot replied that he knew of none who had died.

BOOK REVIEW.

Diseases of the Ear.

By PHILIP D. KERRISON, M. D. 588 pages, 331 illustrations in text, and two full pages in color. J. B. Lippincott Company, Philadelphia and London, 1913. $5.00.

The reviewer has read this book, the latest addition to otologic literature, with a great deal of interest, and regards it, on the whole, as one of the best of the textbooks on otology which the English language has yet produced. The style is pleasing, and the subjects are treated in a full and somewhat discussive fashion. While voicing the writer's own views, it is at the same time judicial and fair to the views of others, and fully recognizes that many procedures in otology are far from having reached finality. The author has the unfortunate habit of using the terms "author," "writer" and the personal pronoun "I" somewhat indiscriminately, and sometimes all three are on the same page; for instance, in two consecutive sentences on page 226 the terms "the writer" and "I know" are used. It seems to the reviewer that the person writing a book exponential of his own ideas should use the personal pronoun "I," and no other, or if adopting the term "author" or "writer" should stick to it.

The importance of the work justifies the consideration of some of its views a little in detail. A very good description is given of the anatomy of the ear at birth, and the changes which take place during the first two years, as well as of the topography of the drum. He denies the statement that the horizontal plate of the squama enters into the formation of the tympanic roof, and says that this is wholly formed by the superior plate of the os petrosum. The mastoid antrum is always present, even in fetal life, and is not a mastoid cell, but the posterior end of the tympanic vault.

The physiology of sound transmission is described not only clearly, but in such a way that it is read with genuine interest. A suggestion is given as to the discovery of malingering, namely, apply the caloric vestibular test whenever total deafness in one ear is claimed. If a quick and normal reaction is obtained, it is probable that the deafness is assumed. The

treatment of furunculosis seems rational, and is given the attention which this common and distressing condition demands. The author regards furunculosis as requiring hospital attention, and finds that patients who are given absolute rest in bed do better than those who are treated as ambulatory cases. A general anesthetic is advised for any aural condition which may require very much manipulation, such as foreign bodies, incision of the drum, and the like. Ten per cent of scarlet fever cases develop tympanic disease. Measles give rise to many cases of acute otitis of severe type, often going on to mastoid trouble. Tinnitus aurium is considered at some length as to its causation, but unfortunately nothing of value is offered as to its treatment. Retraction of the drum is a sign of adenoids in children, and this has not as yet received sufficient attention. Classification of acute middle ear disease is given as acute catarrhal otitis media and acute purulent otitis media, followed by a very good account of the ordinary accepted pathology.

Catheterization is advised during the acute stage of otitis media after drum incision, and he does not believe that there is any danger of infection of the mastoid antrum from this source, as the line of least resistance is through the incised drum. Syringing rather than dry wicking is given the preference in the acute inflammations. In the case of antrum tenderness ice is allowed for twenty-four hours only. The indications as given for operating in acute mastoiditis are safely conservative, neither too hasty nor admitting of dangerous delay. Chronic middle ear suppuration is discussed in a very rational way, from all standponts, and from a personal viewpoint which appeals especially to the reviewer, who is inclined to treat many of his cases of chronic suppurative otitis media conservatively rather than radically. He states: "We should not be slaves to the dogma, too often repeated in otologic literature, that aural discharge is of itself necessarily a danger signal, or one which when persistent calls for operative intervention. If we can satisfy ourselves that the discharge is simply the logical product of exposed mucous membrane or healthy granulation tissue, and that there is no evidence of an active suppurative process involving the bone itself, we should not be too ready to interfere with nature's method of conservation." In certain forms of attic sup-

puration a simple mastoidectomy is advised, in order not to endanger the hearing power, the aditus being very carefully and thoroughly cleared of granulations and diseased bone.

In the section on otosclerosis are given three pictures from Professor Manasse's book, but there is no acknowledgment in the text nor any footnote telling definitely the source from which the illustrations are taken other than in the legend attached to the cuts, nor is there any reference in the text to Professor Manasse's theories. This is due to the fact that Dr. Kerrison's manuscript was in the hands of the printer when Professor Manasse's monograph was issued, and it was too late to make corrections in the text. The cuts were inserted with the permission of Professor Manasse's publishers, and in a later edition a full discussion of his theories will probably be given. On page 253, after epitomizing the symptoms, the remark is made that with such a picture the physician who fails to recognize otosclerosis with stapedial fixation is certainly not a practical otologist. As Professor Manasse has stated there is no symptom complex by which one can with certainty, during life, diagnosticate ostitis metaplastica or otosclerosis, such a statement seems hardly borne out by the facts. An absolutely hopeless prognosis, with reference to the value of any treatment in otosclerosis, is given. The anatomic point is made in the section of the labyrinth that the ordinary description of the scala tympani and scala vestibuli as being respectively the lower and upper cochlea channels is not only incorrect, but confusing, since with the head erect the central axis of the cochlea is not vertical but horizontal, and its direction from the vertical anteroposterior plane of the skull is horizontally forward and outward.

Much space is given to the labyrinth, and an endeavor made to make the whole subject as clear as possible. The rather new term in otologic literature, "arthrodial sense," is used as referring to the sense which tells the blindfolded individual of the position (in flexion or extension) of his joints and through this of his body generally. The reviewer has been unable to find this word, in the sense in which it is here used, in any of the dictionaries at his command, but understands that it is a rather common term in neurologic literature and discussion.

The symptomatology in the chapter on intracranial disease,

and the relation of the ear thereto, is one of the most satisfactory sections, the valuation of each symptom in relation to possible lesions being most carefully considered. He advises in the section on infection of the sinus that the sinus be uncovered whenever there are any symptoms such as to suggest the possibility of sinus infection; that the sinus be exposed whenever any unusual amount of pus is encountered, in order to eliminate the possibility of an abscess in this location; also, whenever there are physical changes present which leave in doubt the question of the health of the sinus groove. No mention, however, is given as to uncovering the sinus in case it should be accidentally injured during the mastoid operation, but only directions as to caring for the hemorrhage. The reviewer is of the opinion that in case of accidental injury to the sinus it would be better to still further bare the sinus, enlarge the nick so as for the moment to produce free bleeding, and then to pack. He believes this is better than to leave a small opening which may, unknown to the operator, carry infection into the general blood stream, whereas, if the other treatment is given and the sinus packed off the risk from this source should be very slight.

In the chapter on brain abscess there is a careful review and study of the individual symptoms and their varying diagnostic value, followed by a most careful study of cerebellar abscess and the symptom relationship between it and cerebral abscess. He cites cases of injury to hearing from cutting of the stapes in paracentesis. Such a case has come within the knowledge of the reviewer.

The various steps of the simple mastoid operation are given in their proper order, and the reasons for each one individually stated. He does not advise removing any but the cortex of the tip, nor the removal of every vestige of diploeic or pneumatic cell; this being not only unnecessary, but physiologically impossible. Nor does he believe that a very smooth or even surface is essential for the formation of healthy granulations; on the contrary, to go further and curette the walls into surfaces of even and polished smoothness may reduce their vitality to a point seriously interfering with normal repair. The blood clot operation is described in the language of its originators, and is not favored. The point is made that in the radical operation the facial nerve is usually injured in its

horizontal rather than its vertical portion, and with the curette rather than with the chisel. In delayed healing in the after-treatment of certain radicals which suppurate longer than is desired, he advises packing with boric acid, very nearly filling the canal, and then completing the filling with gauze, leaving it in for twenty-four hours, and replacing daily. Skin grafting is not advised at the time of the primary operation. He does not think one is ever justified in making definite promises to the individual as to the influence of the radical mastoid upon the hearing. In his own experience the hearing is oftener improved or unchanged rather than curtailed.

In the operative treatment of the labyrinth the various indications and the comparative advantages and disadvantages of the operations of Jansen, Hinsberg, J. D. Richards and the author's own method have been analyzed. Drainage in the great majority of cases is what the labyrinth operation should seek to accomplish. While hitherto an advocate of resection of the jugular vein, he is coming to the conclusion that simple ligation as practiced by the Boston school will answer. Most careful reasoning out of all the steps in all of the operative sections is given, especially that of the lateral sinus operation.

In exploring the dura in suspected brain abscess he thinks it an advantage to make parallel incisions at different points rather than to go diagonally in various directions from an initial point of entrance, as in the case of the latter all of the previous cuts may possibly become infected, whereas in the former method the cuts made which do not reach the abscess cannot be infected by the drainage from the abscess. An ingenious gauze wick for use in drainage is described and illustrated.

Adenoids are regarded as physiologic structures, a point of view which the reviewer has long held, and only when unduly enlarged to be considered as pathologic. Impaired hearing is one of the probable signs of their presence, and granulations along the posterior pharyngeal wall are pathognomonic. He remarks that the forcible use of the curette can remove not only lymphoid tissue, but also muscle tissue with resulting stenosis. Anyone who has examined with the finger postnasally some time after the curette operation for adenoids will be surprised to find how often adhesions of Rosenmüller's fossa to the posterior lip of the eustachian tube remain. He

believes that if in the presence of acute otitis media adenoids are manifestly present, the operation for adenoids should be done at the same time as the drum incision. This can be done with one anesthesia, and many cases of acute ears will not clear up until the nasopharynx is cleared of the adenoid tissue. In considering the question of the relation of aural diseases to life insurance, while recognizing the possible gravity and risk of many lesions, he nevertheless believes that there are many cases in which, in spite of a large perforation allowing a certain amount of aural disease, the character of the lesion is such as to involve little or no risk to life. We have as yet no statistics which are at all reliable as showing the proportion of fatal cases of ear disease to the number of persons afflicted. Even those of the ear hospitals are not reliable, since large numbers of such cases are treated by private physicians, only severe cases coming to the ear hospital or specialist. The subject of artificial aids to hearing is rather slighted, as in most textbooks, a viewpoint which, to the reviewer, seems a mistake, as those artificial aids should be prescribed by the aurist in accordance with the needs of the individual, the same as glasses are prescribed by the oculist.

As showing the rapid advance in otologic science, we have chapters with such titles as "The Value of Salvarsan in Aural Disease," "The Use of Vaccines in Aural Disease," "Aural Disease in Life Insurance" and others, titles which we would have looked for in vain in most of the older otologic textbooks.

Examples of history charts, and the remedies and formulas which the author has found most useful, and a very good index complete the book. The illustrations are good, and add much to the value of the text.

Unfortunately the publisher has seen fit to print the book on paper about the weight of cardboard, making the volume very heavy to hold, and somewhat over four pounds in weight. It could easily have been printed on thinner paper, and would then have occupied not more than half or two-thirds its present bulk, and to the great comfort of the reader. It seems to be the policy of nearly every medical publisher to produce a volume of about the same size and price without much regard to the length of the manuscript. Attention should be drawn to this kind of padding oftener than it is.

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